STUDIES ON THE EFFECT OF POPULATION SIZE AND SELECTION INTENSITY ON ARTIFICIAL SELECTION

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In accordance with the regulations of the University of Edinburgh, I hereby declare that this Thesis has been composed entirely by myself and that all the work described herein was carried out by myself alone, except where otherwise stated.

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SUMMARY

The effects of population size and selection intensity on the selection response in both the short and the long term were examined for two traits in Drosophila melanogaster.

Two independent selection experiments were carried out. Each was set up according to an unequally replicated factorial design incorporating two population sizes (10 and 40 pairs of parents) and three selection intensities (20%, 50% and unselected controls).

In the first experiment, the character under selection was thorax length, and the correlated response in pupae number was examined. The results of this experiment may be summarised as follows:

a) short term responses to selection agreed well with expectations based on heritability estimates from the base population.

b) short term selection response and realized heritability tended to increase with population size.

c) the effect of population size on long-term response was not consistent. However selection response and realised heritability increased with Ni.

d) The behaviour of individual lines suggested a rapid early response followed by a decline in rate of response.

e) Half-lives tended to increase with i and decrease as N increased.

f) Correlated response for pupae number agreed with the direct response. However, the results were not in agreement with the expectations from the estimated base population genetic correlation.

In the second experiment, the character under selection was pupae number and the correlated response in thorax length was examined. The following points were noted:

(a) The early selection response was greater than expected.

(b) The effect of N on short-term selection response and realised heritability was not consistent.

(c) The amount of available nutrients imposed a limit of about 100 pupae per vial. Under these conditions, larval competition resulted in a reduction in larval survival, adult body size and egg production. The time to eclosion was prolonged. The effect of N and i on long-term selection response was obscured by these effects. Correlated response in thorax length was also affected.

There was poor agreement between replicates for both short and long term selection response in both experiments. Standardised correlated responses were highly asymmetrical for these characters.

The means of the two characters were unaffected by inbreeding depression in the first ten generations. The mean pupae number was subsequently seriously affected, while thorax length remained unaffected. Crosses between selected lines yielded heterosis, indicating that differentiation between the lines had occured over the period of selection.

I. INTRODUCTION

Man has learned, through his daily toil, that his work is likely to be more efficient, the better he knows the natural factors involved in it. This continuous struggle is what adds bits of knowledge to that human endowment that has evolved in what is known now, as Science. It provides man with tools to be able to play a better role in nature and grows itself continuously, as it is used in that process.

A good example of what has been said above is the evolution of the scientific concept of effective breeding population size and its use within the context of natural evolution, population genetics and animal breeding.

Perhaps the study of populations entered fully into science with Darwin, as was pointed out by Wright (1967). However, it took time to realize that population size from a genetical view point may be much less than the actual one. When working on how to combine inbreeding, crossbreeding and selection in the most effective way, Wright found that the number of mating individuals is not necessarily equal to the effective number. In his 1931 paper, he described the concept of the effective number (Ne) as two random samples of gametes, N sperms and N eggs drawn from the gametes produced by the generation in question. The population consists of N/2 males and N/2 females, each represented twice from each series of alleles (considering diploids only). He fully acknowledged

in that paper when discussing the reduction of the number of breeding parents due to their unequal progeny contribution, the work by Smith and Calder (1927) on the Clydesdale breed of horses in Scotland in which they found a steady increase in the degree of inbreeding equivalent to that in a population headed by only a dozen stallions.

The knowledge that in a small population gene frequencies can drift a long way apart from their original values due to the random sampling of gametes and that this might explain gene substitutions, made the effect of population size an interesting issue to study, not only from the evolutionary point of view, but within the context of animal and plant breeding.

In his 1938 paper, Wright presents a formula to measure Ne when the number of individuals which mate per each sex are different (of great importance in animal breeding), when are cyclic variations in actual population size and when the offspring contribution of parents varies. Since then, work has been devoted to find ways to measure effective population number under more complicated circumstances and taking into account other factors that affect this measurement (Crow, 1954; Crow & Morton, 1955; Robertson, 1961; Kimura & Crow, 1963; Nei & Murata, 1966; Felsenstein, 1969 and Crow & Kimura, 1971).

The work by Kimura on chance fixation of genes,

using Kolmogorov's equations was a breakthrough that has allowed further studies in small populations in the context of both evolution and animal breeding. Based on that work, Robertson (1960) developed a theory of limits to artificial selection in small populations, that has been advanced by other workers (Latter, 1965b, 1966; Hill & Robertson, 1966; Gill, 1965, etc.). This work has been the basis for a new way of thinking and understanding of new animal breeding schemes and experimental work with laboratory and farm animals (James, J.W. 1972, 1976; Jackson & Turner, H.W. 1972; Rae, A.L. 1974; Eisen, 1974; Frankham et al., 1968a,b; Roberts, 1966a,b).

These new breeding schemes and experimental work have called for statistical research into the estimation of population parameters and their standard errors (Hill, 1971, 1972, 1977; Hill & Thompson, 1977, etc.). This has led to a better understanding of how to apply the basic theory to experimental selection.

The problem of limits to artificial selection in small populations still has several gaps in our knowledge to be filled. Latter (1969) claimed a need for a combined research of computer simulation and experimental work to help in developing models which can be accepted as realistic in the sense of being sufficient to explain the most conspicuous features of response to selection.

We can see clearly from those single examples the

intermingle and feed back of an industry such as animal production with technological and scientific knowledge in the evolution of a concept.

The purpose of this study on the effect of Ne and i (standardised selection intensity) on artificial selection is to add, if possible, information relevant for a better understanding on this interesting and important issue of population genetics.

In selecting Drosophila flies for body length and pupae number in small populations an attempt will be made not basically to check existing theories which predict long term selection events in small populations, but rather more to use them in explaining our results and, with the help of former experimental work, to try to discuss their usefulness and limitations.

The discussion of the genetical constitution of the population I examined for these two rather different traits has been used to contribute to the generalization of our ideas on the genetic mechanisms of quantitative inheritance and in particular of these two Drosophila characters.

II. THE THEORY OF ARTIFICIAL SELECTION

The early theory of artificial selection for a quantitative character has been derived for models in which the population is assumed to be infinitely large. Refinements of the theory have shown that Haldane's approximation (1931) is fairly robust for a few cycles of truncation selection but either as the process advances or the character selected for is influenced by genes of large effect then linear approximation will do its job with rather low accuracy (Griffing, 1960; Latter, 1965a). Kojima (1961) analyzed the effect of truncation selection on the change of genetic parameters in finite populations. He found that his predictions, in which the effect of dominance and the variance of change in gene frequency due to sampling and selection were taking into account, were in agreement with those that would have been expected from the classic theory, provided dominance effects were absent. He also pointed out that the joint effect of finite size of the population and dominance could give rise to a considerable bias in the usual prediction which in the main can be accounted for by inbreeding depression.

Our main concern here is in relation to the prediction of expected limits to selection in finite populations. In this respect, the classic approach has shown both, theoretically (Dempster, 1955) and experimentally (Clayton & Robertson, 1957) to have several limitations. Furthermore, even Griffing's approximation (1960) can be

in great error if the population is small as shown by Gill (1965b). Throughout this section, it will be assumed that there is no natural selection interference of any kind.

A new approach to deal with the problem of small population size was initiated by Robertson (1960). Putting it in his words: "The selection may be expected to increase the frequency of favorable alleles until, in a large population they eventually reach fixation. But if the population size is finite there is a possibility that one allele may be fixed by chance even though there is a more desirable one in the population. The smaller the population the greater will this possibility be". This approach is based in Kimura's (1957) probability of chance fixation of a gene in a finite population.

Let us consider a locus with two alleles A and a with frequencies q and 1-q in a diploid population consisting of a fixed number N of individuals in each generation and let s be the selection coefficient depending on the relative selective advantage of the three possible genotypes at that locus. Robertson (1960) found that the expected gene frequency at the limit (U(q)) is equal to

$$U(q) = q + q(1-q)Ns. \qquad (1)$$

His approach considers U(q) as the proportion of lines in which an individual gene would be expected to be fixed at the limit, as well. N is Wright's effective population size. The total advance expressed as

U(q) - q will be equal to 2N the expected change in the first generation. If 4Ns is much larger than unity but 4Nsq is smaller,than unity U(q) will be about 4Nsq.

For the case of dominance with selection for a recessive gene with frequency q, when the recessive homozygote has selective advantages s, then the chance of fixation is given by

$$U(q) = q + 2/3\ Nsq(1-q^2) \quad \text{approx.} \qquad (2)$$

The total advance will be $2/3\ Nsq(1-q^2)$ which will be similar to that of additive genes if q = .5.

So far, the treatment has been in relation to change of gene frequencies. To put it within the context of artificial selection use is made of Haldane's approximation. For additive gene with a difference of "a" units on the metric scale between the mean of the two homozygotes then $s = a\ i/\sigma$, where i is the superiority of the chosen parents in standard units and σ the standard deviation of the metric character. Using this relation then we can write U(q) as U(q) = f(N i a,q). Assuming no interaction between loci and summing up over loci we will have

$$\Sigma aU(q) = \Sigma af(Nia,q) \qquad (3)$$

This equation tells us that the response to selection depends on the distribution of gene frequencies and effects, on the type of gene action involved and in any population the expected limit of selection is only a function of Ni.

For an additive gene and expressing equation (3) in terms of statistics of measurements of a population, we have

$$R = \Sigma a(U(q)-q) \qquad (4)$$
$$= \Sigma a(q(1-q)\ Ni\frac{a}{\sigma})$$
$$= 2Nih\ \sigma g \qquad \text{for small values of Ni.}$$

Where $\tilde{h}$ is the square root of the heritability and σg the additive genetic standard deviation of the character.

Equation (4) is the usual prediction for response to selection in the first generation times twice the effective population size. For a recessive gene the equation (4) becomes:

$$R = \Sigma a(U(q)-q^2)$$
$$= \Sigma(aq(1-q) + \frac{2}{3}Ni\frac{a^2}{\sigma}\ q(1-q^2))$$
$$= \Sigma aq(1-q) + \frac{4}{3}\ (1+q)\ Nih\sigma g \quad \text{approx.} \qquad (5)$$

The first term is the inbreeding depression.

From equation (4) we can see that the parameters Ni and q play the most important role in guiding our strategies to get the most from a given genetic material.

For small values of Ni the total expected advance is 2N times the change in the first generation and more if recessives at low frequencies are present. Larger values of Ni will yield a larger advance if q is low as the mean value of q(1-q) may well increase during selection as the mean gene frequency increases. In this situation it is possible to have 4N times the change in the first generation. Small values of Ni can only lead to fixation of genes of either large effects or high

frequencies. Only as Ni gets large will there be a good opportunity to fix rare alleles with small effects. When we have either alleles at high frequencies or alleles of large effects and not too low frequencies there is not much advantage in increasing Ni. This agrees with Dempster's comment (1958) that we should keep population size high in storage and in the early generations of selection of non-selected lines.

For a recessive gene causing inbreeding depression a high value of Ni at the beginning may have a two-fold effect. Firstly, if it is at high frequency, by reduction of its frequency, the frequency of homozygotes will be reduced and also as a result its depressive effect on the mean of the character. Secondly, it will reduce its chance of fixation. The lower the rate of inbreeding and the greater the gene effect, then the greater the chance that a harmful recessive will be selected out. Only recessives with small effects will be fixed. Consequently the inbreeding depression will be lower than with a high rate of inbreeding.

Desirable recessives at low frequencies will give more advance than additive ones as a consequence of the increase of the genetic variance within lines up to inbreeding coefficients of .5, as was noted by Robertson (1952).

As the limit is approached asymptotically, therefore it would be meaningless to ask, How long will it take to get there? It is more sensible then, to ask about the

time to get some proportion of the expected advance (e.g. 50%, 90%, 95%, etc.).

Robertson (1960) found for small Ns the half life of an additive gene is of the order of 1.4N. In this context, the half life (L50) is the time to get half way to the limit. For larger values of Ns it was found empirically that as N increases L50 decreases and the lower the gene frequency the more time it will take to reach the half life of the process. Following a different approach, Hill (1969) found similar results for additive and recessive genes. However, a dominant gene will tend to increase L50 at high values of q. At intermediate values L50 will not be much affected by q. Since for a desirable dominant gene inbreeding and selection oppose each other the change in the mean will be negative for low values of Ns. High values of q will give an early reduction in the mean although it will rise later. The larger Ns the sooner the rise will be. However, it will never happen if Ns is low.

So far, the treatment has been concentrated on a single locus model. Now, we will take up the situation in which two or more loci are affecting the character under consideration and they are linked.

In a large population linkage between loci does not affect the ultimate goal of selection whether it be an equilibrium situation (Lewontin & Kojima, 1960) in the case of selection for heterozygotes or fixation of some

desirable alleles (Felsenstein, 1965). What linkage does affect is the rate of advance (Mather, K. & Harrison, B.J., 1949).

In small populations in which chance events may foil the selection goal of fixing all the desirable alleles, linkage disequilibrium generated by sampling of gametes must be taken into account and then, the expected selection limit might be reduced further.

The stochastic treatment of selection for two or several loci in small populations with linkage becomes rather complex. A combination of Algebraic Analysis and Monte Carlo simulations on computers has provided the mean to get a better understanding of the problem.

For a model of two loci as was used by Hill and Robertson (1966) each with two alleles, let the gametes AB, Ab, aB and ab have frequencies f_1, f_2, f_3 and f_4, respectively. Also, let p and q be the frequencies of the alleles A and B and define linkage disequilibrium by the determinant D = f1f4-f2f3, which will be assumed to have an initial value of zero. Assume that these loci have additive selective values r and s at loci A and B, respectively and let c be the recombination fraction between them.

The expected total change in gene frequency of A, U(p.) for low values of Ns assuming that the average heterozygosity declines by a proportion 1/2N each generation and that the average value of D will similarly decline

by a proportion (C + 1/2N) will be given by

$$U(p.) = p. + Nr\,p.(1-p.) + \frac{NsD.}{2Nc+1} \qquad (6)$$

The change in gene frequency is dependent on Nr, Ns, Nc, D and its initial gene frequency.

When dealing with several loci the parameters Nl and n will be needed. Where l is the chromosome length in Centi Morgans and n the loci number (Robertson, 1970).

For the change of the mean of a quantitative character, we use Haldane's approximation to have r = iα and s = iβ, where α and β are the effects of the two loci on the metric character divided by the phenotypic standard deviation.

The change of the population mean will be given by

$$R = \{\alpha(\mu(po) - po) + \beta(\mu(qo) - q_o)\}\sigma$$

$$= 2Nih^*\sigma g^* + \frac{2NiDo\ \alpha\beta\sigma}{2Nc + 1} \qquad (7)$$

where h* and σg* are the square roots of the contribution of the loci to the heritability and genetic variance of the character, respectively. Latter's (1965a) computer simulation runs for a two loci model of equal effect and gene frequency agreed with those expectations. Furthermore, under this situation the presence of epistasis would not alter much the results (Ohta, 1968).

When many loci are involved with free recombination, Kimura's formula (1957) of chance fixation for small Ns

will possibly underestimate chance fixation if the initial gene frequency is low as there will be an increment in p(1-p) by a factor 1/2 due to selection and decrease by a factor 1/2N due to drift each generation. The net outcome would be a temporary increase in the genetic variance. Values of gene frequencies of the favourable alleles at the other loci above .5 will tend to overestimate the expected limit as both genetic drift and selection will reduce the genetic variance.

When the loci involved are tightly linked, linkage will affect gene fixation probabilities. For a two locus model segregation at a locus will affect the gene fixation probability of the other only when its gene frequency is low and its effect is greater than about half of the other. Hill and Robertson (1966) found that for tight linkage to be detectable the gene effect of the locus B should be greater than one half that of the locus A and even when its gene effect was three quarters that of the A, its influence on the chance of fixation of the latter was very small. As the effect of the B locus increases further the chance of fixation of desirable allele in A passes through a minimum and then rises again. This minimum is very dependent on $q_.$. If $q_. \geq .5$ there is almost no influence of tight linkage on the chance of fixation $U(p_.)$.

With many tightly linked loci, we will expect to have the same chance of fixation at the limit as with a

single locus, if the chromosomal effects follow a normal distribution and Nih* is small. When Nih* is high, Robertson (1970) gave an expectation about $3\sigma g^*$. Under this situation, any further increase in Nih* will cause linkage to reduce the expected advance. For values of q. $\neq$ .5 the normal curve will be skewed. Then there will be a tendency for values below .5 to yield a greater advance than expected on the basis of normality and for values above .5 to yield a lower advance.

Low values of q. accompanied by high of Nih* is the situation in which linkage will show its greatest effect. This we saw was observed in the two loci case as well. It is enhanced as n increases.

Loci with unequal gene effects will respond more with linkage than with free recombination. It is due to the fact that genes of small effect will give only a small proportion of their possible gain with free recombination. It was proved to be true at high and moderate values of Nih*.

Variation in the initial gene frequency of the loci will diminish the effect of linkage on possible response. However, high values of Nih* will favour response with free recombination and thus the presence of linkage will reduce the possible response. If some of the genes affecting the character selected for happen to be on different chromosomes, the expected response to selection will be reduced by linkage if there is a variable chromosomal effect.

Now we will look at intermediate values of linkage. When linkage affects the chance of fixation the increase of gene effects will enhance that effect. If gene effects are small the effect of selection on changing gene frequencies is low then the effect of linkage on the expected advance due to selection is less. If the population size is increased there will be higher probability for genes of small effect to keep segregating for a longer period. Then, selection will increase their frequencies and linkage could show its effect. This will hold for dominant loci too (Qureshi & Kempthorne, 1968a).

For high values of q. linkage does not have much opportunity to cause reduction in the chance of fixation. This was found for two loci and for several loci with additive and dominant action. (Latter, 1965b; Hill & Robertson, 1966; Qureshi & Kempthorne, 1968).

For low values of q. linkage will decrease U(p.). It will be a minimum that will depend on the magnitude of the gene effect at the B locus. There is a critical value of q. for the minimum to occur (Latter, 1966 and Hill & Robertson, 1966). Looking at the Figures 2, 3 and 4 of Hill and Robertson, (1966), we can see that the minimum of U(p.) occurs when the effect on the B locus is about double that of the A locus for values of q. $\geq$ .1. When q. $<$ .1 or linkage gets loose the minimum will occur at higher values of NiB. This minimum was found as well for several loci (Qureshi & Kempthorne, 1968 and

Robertson, 1970).

The reduction in response due to linkage was firstly thought to be due to the increase of the chance of fixation of repulsion gametes more frequently than would be expected in the case of unlinked genes.(Latter, 1965b). However, a deeper analysis showed that this decrease is produced by the chance loss of the most favoured gamete AB in many replicates when there is still a segregation period at the gametic frequencies of $f_{11} = 0$, $f_{22} = 0$, $f_{12} = \frac{1}{2} + p$, $f_{21} = \frac{1}{2} - p$. In such a population, the chance of fixation of a repulsion gamete may readily occur before the AB gamete can be recovered by recombination. The tighter the linkage the higher the probability for this latter event to occur (Latter, 1966 and Hill & Robertson, 1966).

The overall outcome from that can be explained as follows. Consider a locus A with a large effect linked to a locus B with small effect. In this situation U(p.) will not be affected by linkage, as the reduction in the chance of fixation of the gamete AB will be balanced by the increase in the chance of fixation of the gamete Ab as linkage tightens. How then does a reduction in response to selection come about? The answer is that although U(p.) is not affected by linkage, U(q.) is. In this case, locus B of small effect is linked to a locus A of large effect and that will reduce the chance of fixation of B. It is due to the fact that whereas the chance of fixation of gamete AB is reduced as linkage

tightens that of gamete aB remains unaltered.

If now we make Niß half the value of Niα then linkage will start showing its effect on U(p.), although small. U(q.) will have its lowest value when Niα is double the value of Niß, then the net effect will be a greater reduction in the response to selection. When NiB is equal to Niα both U(p.) and U(q.) will be reduced by a fair amount. The gamete AB suffers its greatest reduction and the response to selection is at a minimum, as a further increase of Niß will then increase U(q.) without much effect on U(p.) and overall response will rise. This explains the minimum in the Figure 10 of Hill and Robertson (1966).

Response to selection for several loci in the presence of intermediate values of c can be described by Nih*, n, q. and Nl. As n increases, response for high values of Nl will be independent of n and q. and only dependent on Nih* and Nl (Robertson, 1970). Population size proved to be of not much importance if Nih* was higher, otherwise, small N would reduce response. Its effect will be enhanced if c is small. If the gene action shows dominance although we could apply high intensities of selection, the ultimate response will be less than expected if the character shows inbreeding depression and the favoured allele is dominant. For several loci with overdominance tight linkage would reduce the release of

variability even after several generations of random mating. Then, linkage will reduce heterozygosity in small populations even though there is selection favouring the heterozygotes (Qureshi, 1968).

If there is initial linkage disequilibrium less response to selection would be expected than with initial linkage equilibrium. That effect will be larger when $|N\alpha|$ and $|N\beta|$ are large. Ohta (1968) found that with increasing Nc, the effect of $D_{\cdot} \neq 0$ will decrease. This would explain Roberts' (1966c) results when selecting a population coming from the cross of inbred lines of mice after having been mated at random in a large population.

For linkage to act it is necessary that a certain amount of linkage disequilibrium exists, therefore, if a population is in linkage equilibrium when the selection program starts the rate of response in the first few generations will not be affected by linkage. It has been found in simulation studies that it is so. (Latter, 1966; Gill, 1965a; Hill & Robertson, 1966). The extra gain on increasing recombination takes place in the later generations.

Low values of $q_{\cdot}$ and high of $Ni\beta$ will reduce L_{50} as the total response is reduced. Seemingly, linkage starts affecting the selection process after about N generations (Hill & Robertson, 1966) and only for small values of $q_{\cdot}$ linkage will show its effect before $2/ih^*$ generations (Robertson, 1970). If Nih^* and $q_{\cdot}$ are high linkage will

not be able to act as the process is a short one. However, if N and q. are high but ih* is small, the selection process will last long enough for linkage to increase L_{50}. For high values of Nih* the expected value of L_{50} decreases as ih* increases. With dominant effect and tight linkage the rate of response will decrease as N does if i is small but not if it is large (Qureshi, 1968).

Qureshi and Kempthorne (1968) found when they looked at the ratio Rt/Ro (Response at the limit over initial response) that it reduced as i was increased. That is an indication of an increase in early response to the expense of later response. That ratio always was higher for additive genes than for dominant genes as Ro was higher in the former and inbreeding depression may affect Rt. When they fitted a curvilinear model to the change of the population mean, they found that the greater the early response due to high values of i, h^2 and N the greater the curvature was. This can be seen also in Figure 6 of Hill and Robertson (1966).

The variation between replicates of selection programs can tell us about the reliability of our results but it would help us to use the genetic material afterwards as well. Thus, we are interested in knowing which factors are affecting it and what the size of their effect is. The variance of U(p.) is reduced as Ni increases. This reduction is greater with free recombination. With tight

linkage the variance between replicates at fixation is less than would have been expected from their average gene frequency (Qureshi & Kempthorne, 1968).

When Nih* is small, the variation between replicates comes almost entirely from drift, therefore the variance between replicates will be twice the initial genetic variance whatever the tightness of linkage.

When the value of Nih* gets too high the results will be quite different in the two extreme situations of linkage. With free recombination the variance between replicates is almost entirely determined by the mean gene frequency at fixation. When there is no crossing over and n is large the variance between replicates will decline continuously as Nih* increases.

For loci with different gene effects the variance between replicates is generally rather greater than that for loci of same gene effect if Nih* is small but smaller if Nih* is high.

For intermediate values of linkage the variance between replicates will increase as linkage gets tighter if the variance between replicates is higher for tight linkage than for free recombination. If the variance of response yielded by the two extreme situations of linkage are similar, then the variance at intermediate linkage values could be greater. The maximum will be for those values of linkage which give an advance about half way between the two extreme cases.

Following quantitative analysis, formulae for the variance of response to selection, assuming no change in the genetic parameters, have been developed for different situations (Hill, 1971, 1974, 1977a). These can be of great use in designing experiments of selection even if they will last several generations. It seems that the formula to predict variance between replicates when only drift is considered is quite robust and could yield the best estimate of genetic variance between lines (Hill, 1977a).

III. RESEARCH PROGRAMME

Since Robertson's (1960) paper on the limits to artificial selection in small populations, the effect of N has received more attention when explaining outcomes found when lines have been selected long enough to reach a plateau (Roberts, 1966; Verghese & Nordskog, 1968; Eisen, 1972, etc.).

The effect of N and i on limits to artificial selection as a primary objective has been studied by Frankham and colleagues (Frankham et al., 1968 and Jones et al., 1968) and Eisen and colleagues (Hanrahan, et al., 1973; Eisen et al., 1973 and Eisen, 1975).

Those experiments designed to check the theory of selection in small populations have generally found that the parameters N and i play an important role to predict events happening in early and late generations. For instance, it has been found that as N and i increase short and long term responses increase. This was observed by Frankham and colleagues when working with Drosophila abdominal bristles (Frankham et al., 1968a; Jones et al., 1968), Madalena (1970) in sternopleural bristles and Eisen (1975) in his mice postweaning gain research. However, the quantitative expectations for the response at the limit and the half lives of the process were not accomplished in those works. It was argued either they were not at the limit or Robertson's (1960) assumptions were not met in them. Something that should be taken more

into consideration is that N could be very different from its actual value (N*).

Our studies on the effect of N and i on limits to artificial selection tried to look at characters in Drosophila more complex than bristles, to widen the scope of the little experimental information available on this issue. The characters chosen were body length and pupae number which are directly related to growth and reproduction respectively.

This would allow us to encounter different situations in relation to gene action, the relation between genotype and phenotype and relationships of structural and functional characters with fitness. Two independent selection experiments were carried out. In one selection was for body length in upward direction and the correlated response was observed each 5 generations for pupae number. In the other, selection was for pupae number in the same direction and the correlated response was observed each 5 generations for body length. Then, the effect of the changes in one character on the changes of the other at phenotypic and genetic levels could be judged. This should help us to inquire further on the nature of the limit to selection, by assessing the role of factors such as linkage, inbreeding depression, and the correlations between the characters, which have been postulated as possible explanations of the limit (Clayton, 1955; Frankham et al., 1968b; Roberts, 1966b).

of about 50 pairs of parents

The base population was started from a sample drawn from the Dahomey collection cage which has been kept in this laboratory for about 120 generations. From this sample progeny were obtained and distributed in 6 fresh food-containing bottles of half pint size. They were allowed to mate and lay eggs avoiding overcrowded conditions. Each time a new bottle was initiated flies from at least 3 bottles were introduced in it to mate and lay eggs. Preference was always given to bottles with newly emerged flies. This large population made out of flies in the 6 bottles was maintained throughout the research program and it is called the large base population. Any time a comparison in a trial was performed, flies from these 6 bottles were used as controls.

Two generations after the initial sample was placed under these conditions the lines involved in these experiments were started. This was done gradually to have them randomly distributed over 15 days, the period necessary for handling them all. Then, we had a generation interval throughout this experimental program of that length of time. Flies were always kept in a room at 25^{o}C and fed with the standard food used in this laboratory.

Our selection experiments were set up as an unequally replicated factorial design of two population sizes (10 and 40 pairs of parents) and three levels of selection intensity (100%, 50% and 20%). They were arranged as shown in Table 1.

TABLE 1. Experimental design, treatment code designation*, numbers of replicates per treatment (λ) and total number of pairs scored each generation in each replicate (T/2).

Population size (pairs of parents)		SELECTION INTENSITY (P)		
		20%	50%	Controls
10	Code	BSH	BSM	BSC
	λ	4	4	4
	T/2	50	20	10
40	Code	BLH	BLM	BLC
	λ	2	2	2
	T/2	200	80	40

*B stands for body length.
S stands for small population size.
L stands for large population size.
M stands for medium selection intensity.
H stands for high selection intensity.
C stands for control population.

The values of N* and i were in the range of those of Frankham and colleagues and Eisen.

Estimation of heritabilities of both characters and the genetic and phenotypic correlation between them was carried out in the base population at the beginning of the program and for each line at generations 5, 10 and at the limit. For these estimations, and their standard errors, the methods of offspring on parent regression of Hill (1970) and Reeve (1953) were used.

From samples of the base population two progeny tests were carried out. In the first, body length of 200 females and 200 males was measured. Then the largest 20 females and 20 males and the smallest 20 females and 20 males were selected and assortatively mated in individual vials. From each vial 5 females and 5 male progeny were measured for body length and 5 females were put into fresh food-containing vials to assess the number of pupae produced. Regressions of mean body length of progeny (y) on mean body length of parent (x), (byx) and mean pupae number of female progeny (w) on mean body length of parents (x), (bwx) were calculated.

From the measurements of the 200 parents, mean and variance of body length were estimated. The two hundred females were all allowed to lay eggs to assess their pupae production and then to correlate it with their body length in order to get the phenotypic correlation of body length with pupae number (rp).

We use byx as an estimate of the heritability of body length.

In the second progeny test, pupae number of 200 females was counted and the 20 with the largest and the 20 with the smallest values were selected and 5 males and 5 female progeny of each were measured for body length. The 5 female progeny of each selected vial were put into a fresh food-containing vial to assess their pupae production. From the counting of pupae number of the 200

females, mean and variance of this trait were estimated.

Regressions of mean pupae number of female progeny (w) on pupae number of mother (z), bwz and mean body length of progeny (y) on pupae number of mother (z), byz were calculated. We used bwz as an estimate of half the heritability of pupae number.

The genetic correlation (rg) was estimated using the formulae

$$rg = (byz\ bwx\ b^{-1}_{yx}\ b^{-1}_{wz})^{1/2}.$$

Four brother x sister mating lines were developed along with the selection experiments and were called inbred lines (I).

The selection on each line was stopped when almost no response was observed for several generations and the phenotypic variance was nearly exhausted. Then two generations of relaxation followed. Some lines were chosen and crossed before relaxation. In making these crosses, inbred lines were involved too.

The lines selected for thorax length are generally called body lines and those selected for pupae number, pupae lines.

IV. SELECTION FOR BODY LENGTH

1. Introduction

Artificial selection is based on phenotype but with the intention of changing the genetic structure of the population. Therefore, we will always be interested in having a better knowledge of the character selected for at the genotypic level. Then, when checking a theory of artificial selection it is interesting not only to know how it explains the results obtained, but what the architecture of the genetic variation of the character selected for looks like as well. For this reason, the choice of characters to work with should be made in the way to meet situations which will give us a wider test of the theory, taking into account the level of our present knowledge.

Body length in Drosophila has been used as a character to select for as it is a good indicator of growth. Thorax length, measured from the tip of the scutellum to the anterior edge of the thorax as described by Robertson and Reeve (1952) was the actual measurement performed. It is closely correlated to body length and it is very convenient since it does not change after 4 hours after the fly's emergence or with any position a fly takes when etherised to be measured. Thorax length has shown to have a heritability, h^2, of about .35 (Tantawy, 1951; Robertson & Reeve, 1957; Latter & Robertson, 1962). Inbreeding does depress the mean thorax length when it gets to high levels after increasing rapidly. (Tantawy, 1957; Robertson & Reeve, 1952). However, the latter workers did not find

inbreeding depression in one of their lines and Tantawy had very little inbreeding depression for a double cousin mating system at a 74.8% level of inbreeding; nevertheless, these lines showed significant heterosis when crossed. Body weight did not show significant inbreeding depression (Kidwell & Kidwell, 1966; Martin & Bell, 1960; Frahm, 1965).

Selection on body weight or body length has given response in the early generations as expected from the parameters in the base population. However, Frahm (1965) had higher realized heritabilities than expected, but Robertson and Reeve (1952) did not find significant response in the first 20 generations of selection. Long term responses have been poor and erratic. Frahm's lines rapidly stopped improving. Robertson and Reeve (1952) had no response in the first 20 generations of selection. Then, response started accompanied by an increase in within line variance. That response, however, did not last very long for their short-thorax line, although the long-thorax line kept responding up to about generation 45. They gave as a possible explanation for this peculiar behaviour, the magnification of the effect on the character of some genes, which can not be fixed, due to selection of modifying genes.

2. Materials and Methods.

2.1 Genetic material and its handling.

From the base population generated as explained in the previous chapter a sample of 200 females and 200 males were measured to have an idea of the shape of the frequency distribution of thorax length and to estimate the phenotypic and genetic parameters. For the females, the number of pupae was counted too. From Figure 1, we can see that the frequency distribution of body length is not far from normal, with a mean of 91.8 (1/100 mm) for females. To draw the figure only measurements of females were included. As the variance in each sex is very similar in Table 2 a pool variance of both sexes is presented. This population has a smaller thorax length mean than that reported by Robertson and Reeve (1952) but shows more phenotypic variance. The heritability for this trait is much smaller than the values reported by Tantawy (1951), Robertson and Reeve (1957) and Latter and Robertson (1962).

The phenotypic correlation between thorax length and pupae number is not significantly different from zero, although there is a negative tendency. Tantawy and Vetukhiv (1960) found in Drosophila pseudoobscura a highly positive correlation of thorax length and egg production, whereas there was no correlation between the former and egg viability. Robertson and Reeve (1957) gave an average figure of .235 for the correlation between thorax length and egg production. The same authors found a

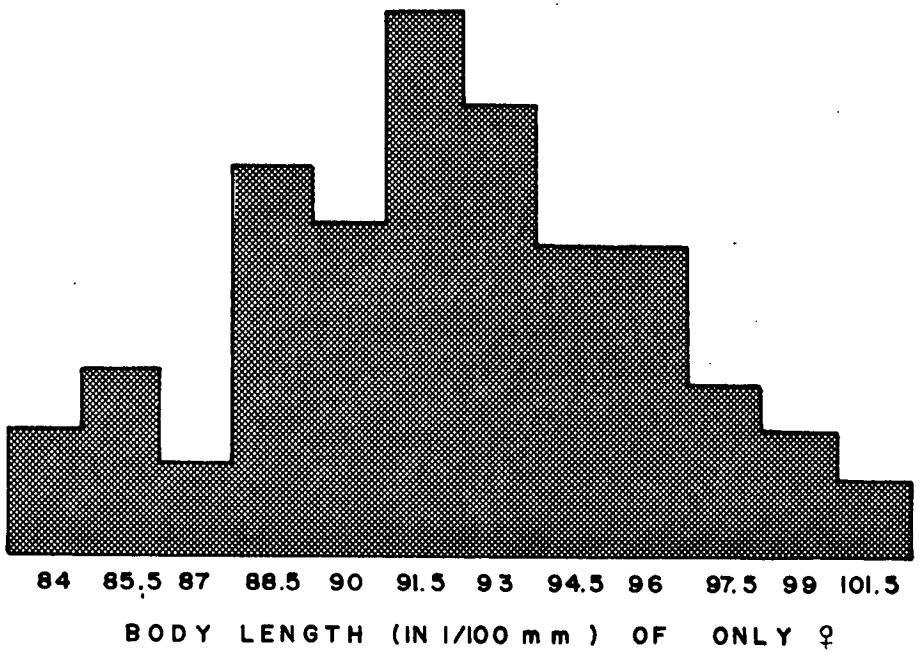


FIG. 1 FREQUENCY DISTRIBUTION OF BODY LENGTH

(BASE POPULATION)

TABLE 2- GENOTYPIC AND PHENOTYPIC PARAMETERS OF BODY LENGTH IN THE BASE POPULATION

n	MEAN ♂	MEAN ♀	PHENOTYPIC VARIANCE (♂ and ♀)	h^{2}*	r_p**	r_g***
200	79.07 ± 32	91.8 32	20.72	.143 ± .03	-.054 ± .07	-.958 ± .10

h^{2}* HERITABILITY OF BODY LENGTH, r_p** PHENOTYPIC CORRELATION OF BODY LENGTH WITH PUPAE NUMBER,

r_g*** GENETIC CORRELATION.

positive genetic correlation of .13 between those characters. I found a phenotypic correlation of -.054, which was not significantly different from zero and a peculiar genetic correlation of -.958 which was statistically significant (see Chapter 3 for details of genetic parameters estimation).

Each line was kept in a single half-pint bottle. Only the large lines were kept in two bottles, taking care to randomize the introduction of parents into them and the collection of offspring. Once emergence started in a bottle, flies were taken out. Early in the morning, all the flies which had emerged the night before were thrown away, then collection of flies was made at 6 hour intervals during the day, until the number of males and females needed were obtained. After collection, flies were sexed and kept separated in fresh-food containing vials until the day of measurement. All the time the flies were kept in a room at 25°C except when they were being sexed and measured for selection. Mass selection was practiced keeping each fly in an individual empty vial, since its measurement until selection was made. This never lasted more than 4 hours. Selection was carried out in both sexes. As soon as the males and females were selected they were put into a half-pint bottle containing fresh food and returned to 25°C to mate and lay eggs for about 12 hours. The idea was to avoid overcrowding but to ensure enough flies for the next generation.

2.2 Estimation of selection response and its analysis.

The selection response will be presented in several ways. As absolute values, deviations from the mean of controls of the same population size and deviations from the mean of the large base population. When drawing graphs, the selection response was plotted against generations. Regressions of response taken as deviations from large base population on generations and on selection differentials up to generation 10 and then at 5 generation intervals will be presented. Regression coefficients of absolute response on generation number up to generation 10 were used to carry out an analysis of variance, whose results were discussed in the short term response to selection. The total response was calculated as the deviation of the mean of the observed values when the lines were plateaued from the mean of the large base population. This was used to compare with Robertson's (1960) predictions of limits.

2.3 Estimation of genetic parameters.

In generations 5, 10 and at the limit, estimation of heritability for body length and pupae number, and phenotypic and genetic correlations were estimated for each line. Offspring on midparent regression was carried out following Reeve's (1953) and Hill's (1970) methods for body length and daughter on mother regression for pupae number. In each case 50 pairs of flies were measured and the longest and shortest 10 pairs were selected and

assortatively mated; each pair in one fresh food containing vial. Three male and three female offspring were measured. Correlated responses were observed each five generations and at the limit. From them, realized genetic correlations were calculated.

3. Results.

3.1 Short term responses.

Short term selection response refers in this work to the change in the population mean observed in the first 10 generations of selection. Table 3 shows the expected response in the first generation estimated from the parameters of the base population. Comparing expected values to the observed ones, presented in the same table, we can see that the values for lines of intermediate intensity of selection are in fair agreement with expectations. However, those for lines of high selection intensity are smaller than the expected ones. $\overline{BLH}$ was slightly higher than $\overline{BSH}$ but quite superior to $\overline{BLM}$ and $\overline{BSM}$. $\overline{BSH}$ in turn exceeded $\overline{BLM}$ and $\overline{BSM}$. In general there was great variation between replicates for all treatments, a common feature in this sort of experiment (Clayton et al., 1957; Frankham et al., 1968, etc.).

All selection treatments yielded response to selection in the first generations (see Fig. 2). Small population size treatments started with larger means than large population size ones, however, at generation

TABLE 3 Response to selection for body length (expressed as regression coefficient of accumulative response on generation number) and expected values.

BSM_1	.357 ± .02	BSH_1	.617 ± .08
BSM_2	.453 ± .02	BSH_2	.436 ± .10
BSM_3	.249 ± .04	BSH_3	.300 ± .11
BSM_4	.289 ± .04	BSH_4	.424 ± .12
$\overline{BSM}$	.328 ± .05	$\overline{BSH}$	.409 ± .07
$\widehat{BSM}$	310	$\widehat{BSH}$	.600
BLM_1	.466 ± .20	BLH_1	.362 ± .10
BLM_2	.203 ± .09	BLH_2	.696 ± .20
$\overline{BLM}$	.352 ± .15	$\overline{BLH}$	.465 ± .13
$\widehat{BLM}$	.350	$\widehat{BLH}$	.630

BSM = Lines of small size (S) and medium intensity of selection (M), selected for body length (B).

BSH = Lines of small size and high intensity of selection (H), selected for body length.

BLM = Lines of large size (L) and medium intensity of selection, selected for body length.

BLH = Lines of large size and high intensity of selection, selected for body length.

The cap symbol stands for expected value.

The expected values were calculated using parameters of the large base population. The accumulative respon e is expressed as deviations from the average of large population size control ($\overline{BLC}$).

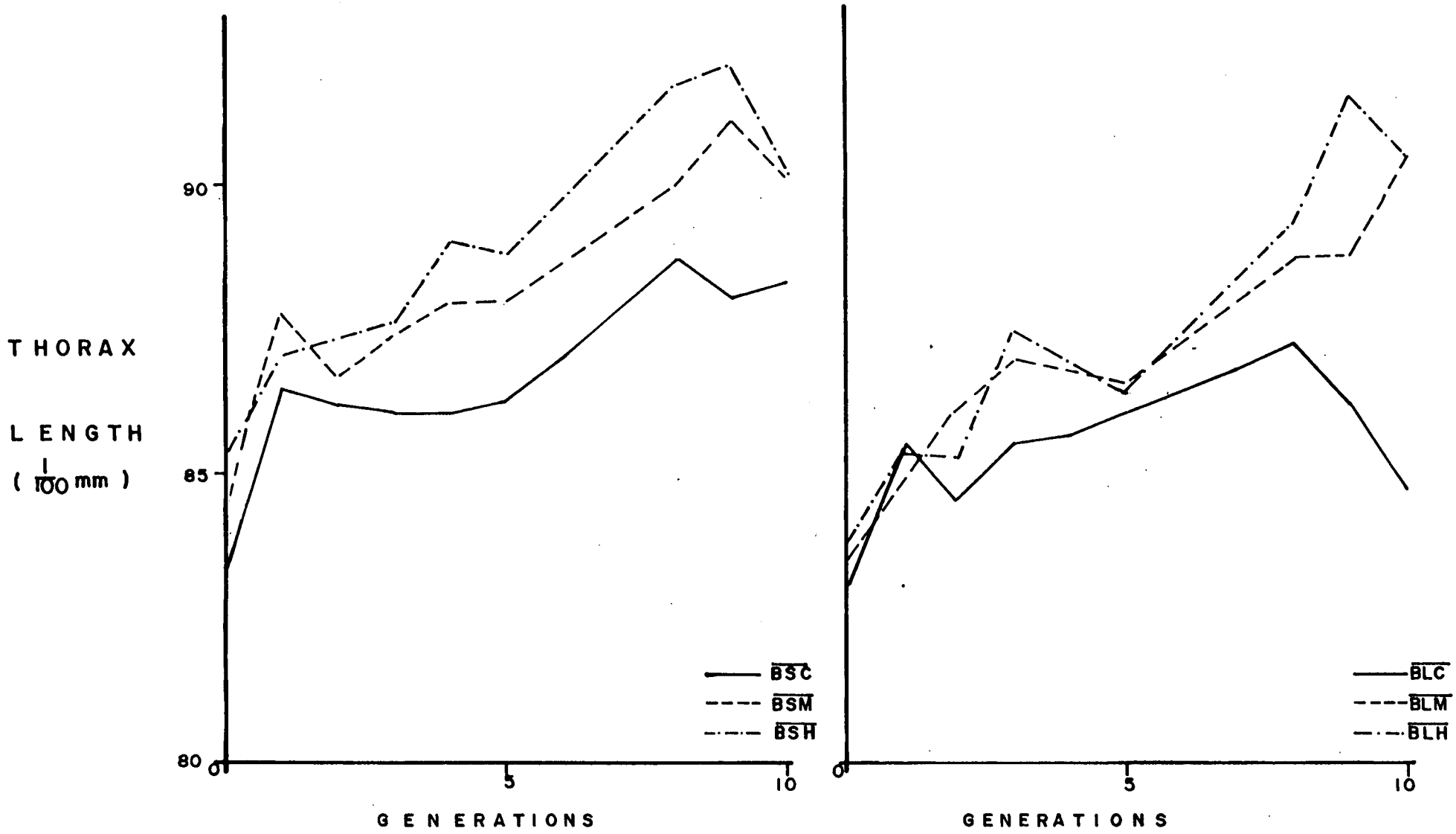


FIG.2 GENERATION MEANS AVERAGE OF BODY LENGTH OF THE $\overline{BSC}$, $\overline{BLC}$ - $\overline{BSM}$, $\overline{BSH}$, $\overline{BLM}$ AND $\overline{BLH}$ LINES. IN THE 10 FIRST GENERATIONS

ten, the four selection treatment means were much the same. An early rise was observed even in controls. After that a steady increase followed in the selected line averages. In general the response was linear and any departure in some lines must be due to environmental factors (see BLH_1 and BLM_2 in Fig. 3c). $\overline{BSC}$ was almost constant from generation one to five, but then kept rising at a steady rate as selected lines. $\overline{BLC}$ after increasing for the first eight generations to a low steady rate had a sharp decline in generations nine and ten.

Figure 3 presents generation means of body length of individual replicates. There is a five generation period of steady increase in which the replicates are much the same. There is, however, more divergence within the treatments with higher selection intensity. Then, a two generation period of noise due to a shortage of food in our laboratory made the replicate means show great variation.

Figure 4 shows control replicates of small and large population size. It is notable that there was a steady increase of the mean of replicates of small size in the last 5 generations. BSC_1 was the most erratic of the four. Large population size replicates showed much less tendency to rise, but differed in their behaviour. Whereas, BLC_1 had risen steadily all the period but the last two generations BLC_2 kept much unchanged for the last seven generations.

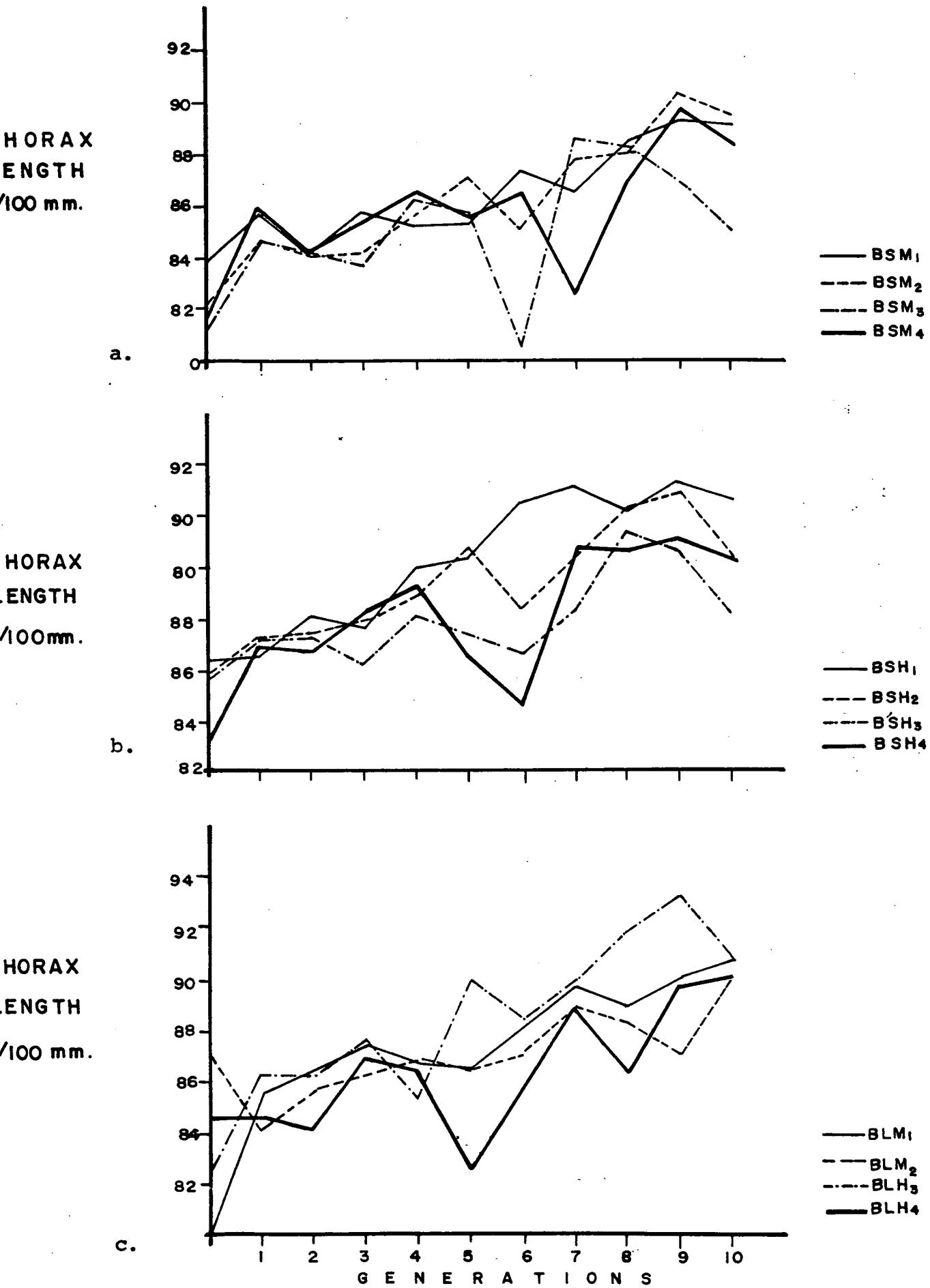


FIG. 3 GENERATION MEANS OF BODY LENGTH OF INDIVIDUAL REPLICATES OF THE BSM, BSH, BLM AND BLH TREATMENTS

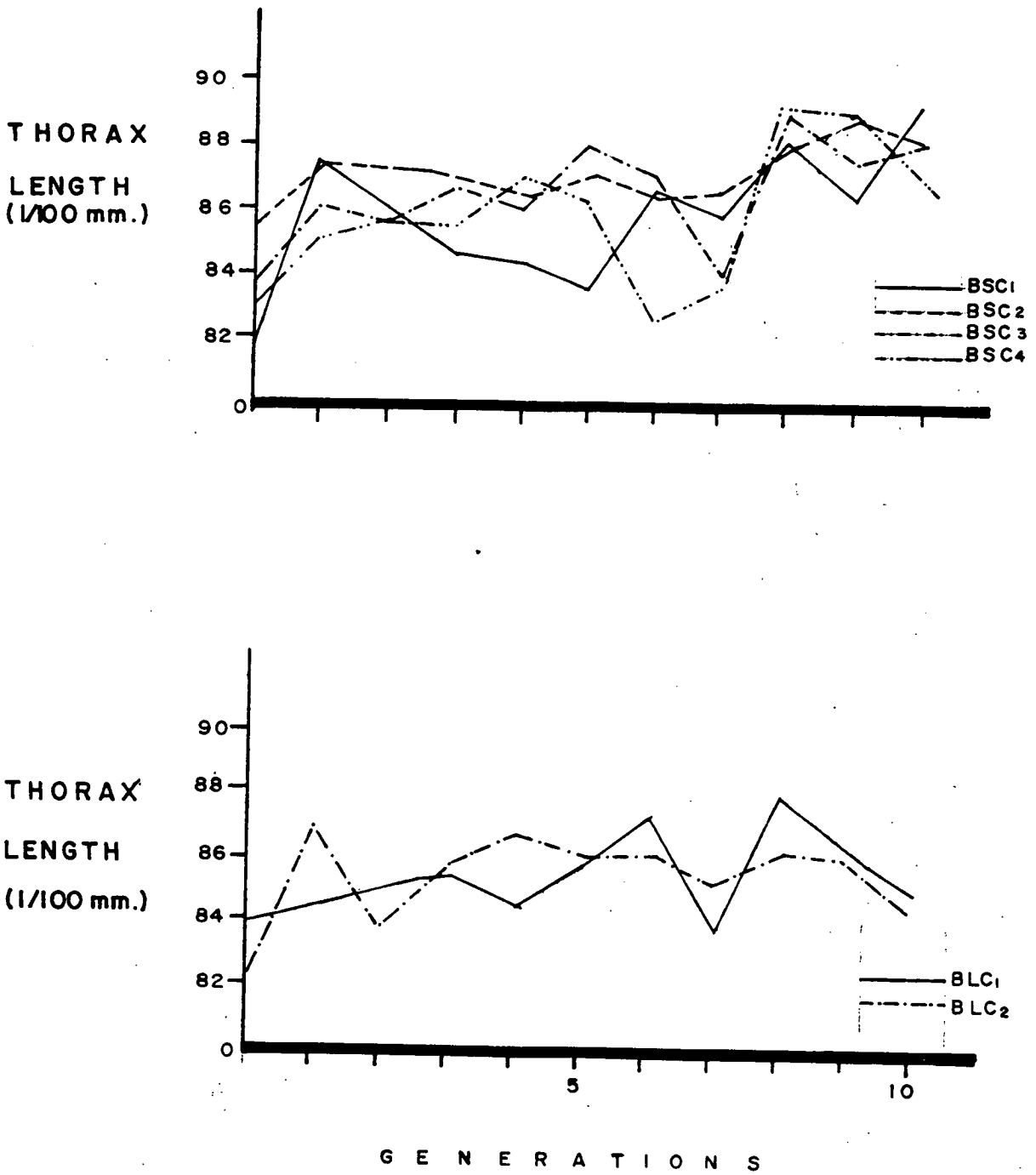


FIG. 4 GENERATION MEANS OF CONTROL BODY LENGTH REPLICATE LINES

Table 4 gives an average picture of the whole short-term selection period. It is obvious that the increase of small population size controls is reflected in the $\overline{BSC}$ value of .314. However, the value of .159 for $\overline{BLC}$ is a consequence mainly of the early increase. That difference of $\overline{BSC}$ and $\overline{BLC}$ can be attributed to drift. Here again, the variation between replicates of the same treatment is quite remarkable.

Taking the regression coefficients of Table 4, as variates an analysis of variance was carried out. It is presented in Table 5. No significant effect of population size was found although the tendency was to have more response in large populations. The selection intensity was a significant factor and the comparison between control and selected lines was highly significant (contrast 1). However, when comparing lines of high selection intensity with lines of intermediate selection intensity, the difference was not significant (contrast 2). The tendency was to have more response as i increased within N. The effect of N between selected lines was not significant (contrast 3).

Large variation between replicates is a common feature of selection experiments. Our data are no exception. However, replicates of BSM treatment (small population size and intermediate selection intensity) had less variation than the others (see Fig. 3 and Table 4). That is not our expectation. In BSH treatment, replicate BSH_3

TABLE 4. Regression coefficients of accumulative response to selection for body length on generation number of control and selected lines and replicate averages.

BSC_1	.403 ± .17*	BSM_1	.523 ± .08**	BSH_1	.776 ± .08**
BSC_2	.190 ± .07*	BSM_2	.612 ± .10**	BSH_2	.595 ± .10**
BSC_3	.306 ± .13*	BSM_3	.409 ± .22*	BSH_3	.372 ± .12**
BSC_4	.358 ± .19*	BSM_4	.450 ± .17*	BSH_4	.583 ± .18**
$\overline{BSC}$	.314 ± .14	$\overline{BSM}$	.498 ± .14	$\overline{BSH}$	.581 ± .12
BLC_1	.195 ± .11	BLM_1	.788 ± .15**	BLH_1	.520 ± .16**
BLC_2	.123 ± .13	BLM_2	.358 ± .10**	BLH_2	.836 ± .15**
$\overline{BLC}$	.159 ± .12	$\overline{BLM}$	.573 ± .12	$\overline{BLH}$	.678 ± .15

BSC = (N = 20, P = 100%) small size control of lines selected for body length.

BSL = (N = 80, P = 100%) large size control of lines selected for body length.

The other symbols as in Table 3.

* ($P < .05$) ** ($P < .01$)

TABLE 5. Analysis of variance of the regression coefficients of Table 4, showing the effect of population size and selection intensity on response to selection.

Source of variation	d.f.	Mean square
Population size (N)	1	.0001
Selection intensity (i)	2	.1995**
N x i	2	.0258
Error	12	.0229
Mean comparisons (linear contrasts)		
(1) 2 $(\overline{BSC} + \overline{BLC}) - (\overline{BSM} + \overline{BSH} + \overline{BLM} + \overline{BLH})$		.5722**
(2) $(\overline{BSM} + \overline{BLM}) - (\overline{BSH} + \overline{BLH})$		.0942
(3) $(\overline{BSH} + \overline{BSM}) - (\overline{BLH} + \overline{BLM})$		.1715

$i = \frac{z}{p}$ = (where z is the normal ordinate at the truncation point) is the standardized selection differential and it will be used as a measure of selection intensity.

** ($P < .01$).

had a very low value compared to the others and something similar happened in BLH treatment with BLH_1. There can be seen as well the great difference between replicates of BLM treatment.

Another point that should be considered in short-term selection response, and perhaps, as well when studying limits of selection is the initial sampling. A difference between replicates of about .5σ can be seen in Figure 3 (a and b). In Figure 3c, BLM replicates differ by more than one σ. Although it might well be an environmental effect, genetic sampling can not be ruled out.

Realized heritabilities for the first 10 generation are presented in Table 6. There the line means were taken as deviations from controls of same size and from the base population. For the former, small lines showed smaller values than expected, whereas BLH lines had an average near the expected value and BLM lines one higher than that. When the responses were taken as deviations from the large base population all the values of $\hat{h}^2$ increased. These values were higher than the heritability estimate in the base population. In these $\hat{h}^2$ estimates, we see that as N increases $\hat{h}^2$ increases too, but for i the outcome is the other way around. This is due to poor performance of BSH_4 line in lines of small size and BLH_1 in lines of large size.

TABLE 6. Realized heritabilities ($\hat{h}^2$) for the 10 first generations†

	1	2		1	2
BSM_1	.18 ± .11**	.26 ± .11**	BSH_1	.19 ± .04**	.25 ± .04**
BSM_2	.19 ± .11**	.23 ± .11**	BSH_2	.09 ± .04**	.15 ± .04**
BSM_3	.03 ± .11	.14 ± .11	BSH_3	.13 ± .04	.12 ± .04
BSM_4	.05 ± .11	.16 ± .11*	BSH_4	.06 ± .04*	.13 ± .04**
$\overline{BSM}$	.07 ± .05*	.19 ± .05**	$\overline{BSH}$	.08 ± .02*	.15 ± .02**
BLM_1	.22 ± .06**	.27 ± .06**	BLH_1	.12 ± .03*	.13 ± .03**
BLM_2	.20 ± .06**	.24 ± .06**	BLH_2	.19 ± .03**	.21 ± .03**
$\overline{BLM}$	.21 ± .04**	.24 ± .04**	$\overline{BLH}$	.16 ± .02**	.18 ± .02**

1 $\hat{h}^2$ as deviations from $\overline{BSC}$ and $\overline{BLC}$

2 $\hat{h}^2$ as deviations from base population mean.

† The standard errors were calculated according to Hill (1972).

* $P < .05$ ** $P < .01$

3.2 Long term responses.

Although artificial selection aims at changing the mean in one direction there are other factors which may result in variation of response even to the extent of reversing the change in mean. However, experimental responses to selection show some characteristics in common which will allow us to eventually make generalizations of the genetic dynamic structure of Mendelian populations. Falconer (1955) gives an enlightened discussion about this topic. One of these generalizations is that the phenotypic variance remains constant during the selection programme (Robertson & Reeve (1952) selecting for body length in Drosophila; Jones et al. (1968) selecting for abdominal bristle in Drosophila; Eisen (1972) for litter weight and (1975) for postweaning growth in mice; Bakker et al. (1978) for litter size in mice). However, the rise of the phenotypic variance in later generations allowed Robertson and Reeve (1952) and Frankham et al. (1968b) to achieve further response as they were able to exert higher selection pressure. In all our selected lines it was observed that there was an early sharp decrease in variability which was reflected in the reduction of selection differentials, but after about the 10th generation the phenotypic variability tended to remain more or less constant (see Figs.,1, 2, 3, 4 and 5 in Appendix).

Large population size selected lines showed less oscillation of their phenotypic variance between generations

than small population size lines and kept more of their phenotypic variance. BLC lines showed great oscillation in their variance, however, it remains much the same as average of the whole period. Phenotypic variance went down rather quicker in small population size controls in the first 10 generations and then tended to remain the same in average thereafter.

The cumulative selection differential in the selection lines was of the order of about 16σ for the BSM and BLM lines and 30σ for the BSH and BLH lines (σ is the phenotypic standard deviation in the initial populations) (See tables 1 and 2 in Appendix). The BSH lines had the largest cumulative selection differential of about 32σ in average. It can be seen from Table 7 that BLH lines with less cumulative selection differential ($\Sigma\delta$) than BSH lines gave more, or about the same, response. An explanation for that is that 3 replicates BSH were showing almost no further response after generation 27 and however were kept along with the BSH_2 which was still improving.

Total responses shown in Table 7 are presented as deviations from controls of same size and as deviations from the large base population. It should be pointed out that whereas all BSM and BLM had reached a plateau the BSH_2 of BSH and both BLH lines, perhaps could yield further improvement (see Figs. 8 and 9). The first set of values are much smaller, as controls of same size increased their mean throughout the selection program.

TABLE 7. Effective population size, expected responses at the limit, actual responses and accumulative selection differentials of body length selected line averages.

Lines	N[a]	Ni	RL[b]	RL_+^c	RL_o^d	$\Sigma\delta^e$	$RL_+/\Sigma\delta$
BSM	12	9.20	7.4	6.5±.58*	11.0±.58	50.78	.128
BSH	12	16.46	14.4	9.6±1.10	14.8±1.10	101.99	.094
BLM	48	37.92	33.6	6.9±.40	8.8±.60	49.54	.139
BLH	48	66.72	60.4	11.6±.40	13.8±.60	92.55	.125

a Effective population size, equal to actual value times .6, following Crow (1954).

b Expected response at the limit, equal to 2NR., for an additive model, Robertson (1960).

c Observed response at the limit as deviation from own control.

d Observed response at the limit as deviation from the large base population.

e Accumulative selection differentials.

* For the standard errors of the observed responses pool estimated obtained from the actual between replicates variance and $t\frac{Va}{N}$ values were used.

The values are close to the prediction of Robertson (1960) for small values of Ni. It is quite remarkable how as Ni increases that expectation is more in disagreement with the results. As i increased within N, response to selection increased. Increasing N within i tended to give a higher response for RL+, but not for RLo in which small populations tended to yield greater response. But if we take into account that BSH and BSM lines had greater cumulative selection differentials for having more generations of selection than BLH and BLM I would say that as N increased, the response to selection tended to increase for a given amount of selection applied. This can be seen from the last column of Table 7.

Variability among replicates was interesting (see Table 8). The BSM lines with small population size and low intensity of selection varied less than the BSH, BLM and BLH lines. BSH lines had the greatest variability. The outstanding performance of BSH_2, which showed greater response than the other BSH lines contributed a great deal of this between replicate variance.

Large population size lines of both selection intensities showed about the same variance. For one or another reason variability among replicates seems to be a common feature of selection experiments. Population size causing genetic drift on its own can not explain our results in that respect.

To attach a standard error to the response averages I used t Va/N to estimate the variance of response following

TABLE 8. Responses at the limit (as deviations from own controls and base population) and cumulative selection differentials of body length selected lines and replicate averages.

	RL+[a]	RLo[b]	Σδ[c]		RL+	RLo	Σδ
BSM_1	6.9 ± 1.9	11.1 ± 1.7	47.2	BSH_1	8.7 ± 2.0	13.6 ± 2.0	106.3
BSM_2	5.8 ± 1.9	10.3 ± 1.7	50.9	BSH_2	13.9 ± 2.0	19.3 ± 2.0	100.9
BSM_3	6.7 ± 1.9	11.5 ± 1.7	48.3	BSH_3	7.5 ± 2.0	12.8 ± 2.0	101.1
BSM_4	6.6 ± 1.9	11.2 ± 1.7	51.3	BSH_4	8.4 ± 2.0	13.5 ± 2.0	100.1
$\overline{BSM}$	6.5 ± .21	11.0 ± .23	50.7	$\overline{BSH}$	9.6 ± 1.1	14.8 ± 1.1	101.9
BLM_1	7.7 ± .9	10.4 ± .9	48.3	BLH_1	10.8 ± .9	12.2 ± .9	91.5
BLM_2	6.1 ± .9	7.2 ± .9	50.7	BLH_2	12.4 ± .9	15.3 ± .9	93.5
$\overline{BLM}$	6.9 ± .6	8.8 ± .6	49.5	$\overline{BLH}$	11.6 ± .6	13.8 ± .6	92.5

a Response at the limit as deviation from own control.

b Response at the limit as deviation from base population.

c Cumulative selection differentials.

The standard errors were calculated using t Va/N as proposed by Hill (1974), and for the averages a pool estimate using the actual between replicates variance and Hill's (1974) proposed estimate was used except for $\overline{BSM}$ in which the actual value was used.

Hill (1974) and a variance calculated from the actual values. As they did not differ too much I pooled them in making the calculation. It was not done for $\overline{BSM}$ as Hill's estimate was rather greater than the actual one.

The pattern followed by the selected lines can be seen from Figures 5 and 6. Table 9 would help as well to show that pattern.

$\overline{BLH}$ lines after a high early rate of response slowed down after generation 15. At generation 10 differentiation between $\overline{BLM}$ and $\overline{BLH}$ lines started (see Figs. 5 and 6). BLH_1 and BLH_2 came to be differentiated over the early generations.

$\overline{BLM}$ lines reduced their rate of response from generation 10 onwards. BLM_2 line even had a negative response in the last period (see Table 9). BLM_1 line rose at generation 25 and started to be differentiated from BLM_2 (see Fig. 9).

$\overline{BSM}$ lines had a great increase of response between generation 10 to 20 and then slowed down a little in later generations to reach a plateau about the 26th generation. However, BSM_2 replicate which was the best in the early generations slowed down drastically between generations 10 to 20 and kept at a low rate of response thereafter (see Table 9).

The rate of response of $\overline{BSH}$ lines was steady all the way through. BSH_1 and BSH_2 lines showed high rates of response in the early and last period. This pattern of response was observed as well by Robertson and Reeve (1953)

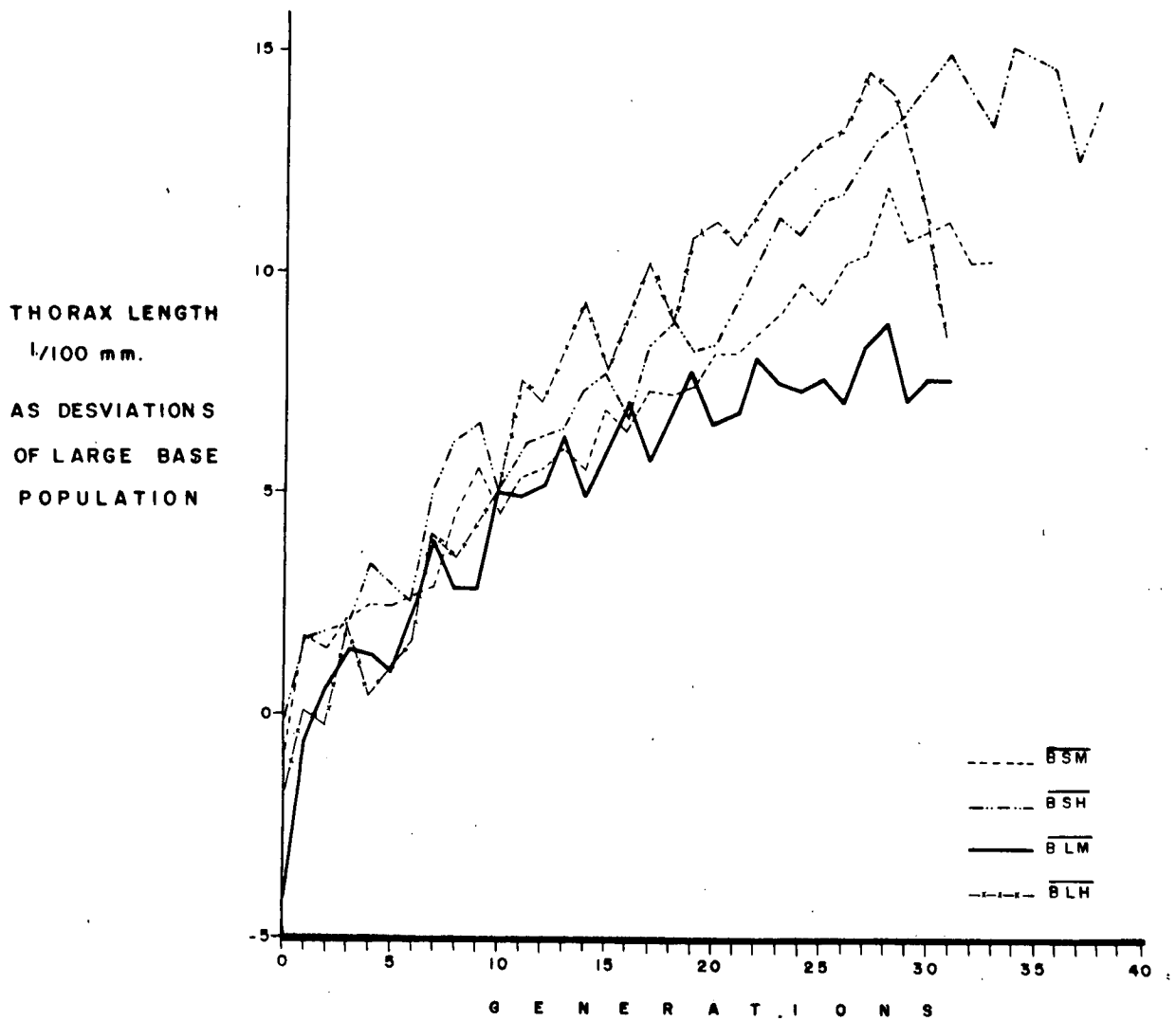


FIG. 5 SELECTION RESPONSES OF BODY LINE AVERAGES

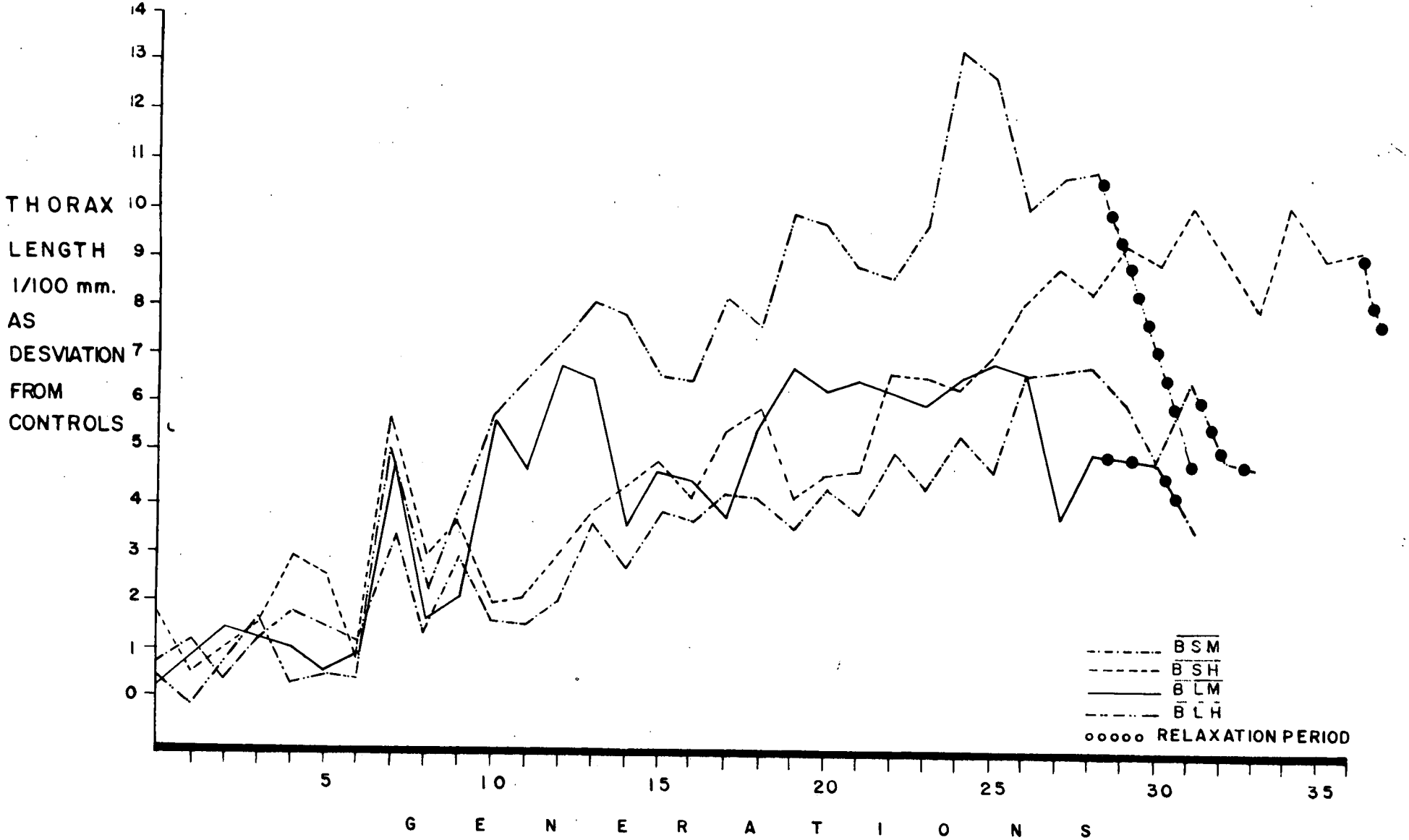


FIG. 6 SELECTION RESPONSES OF BODY LINE AVERAGES

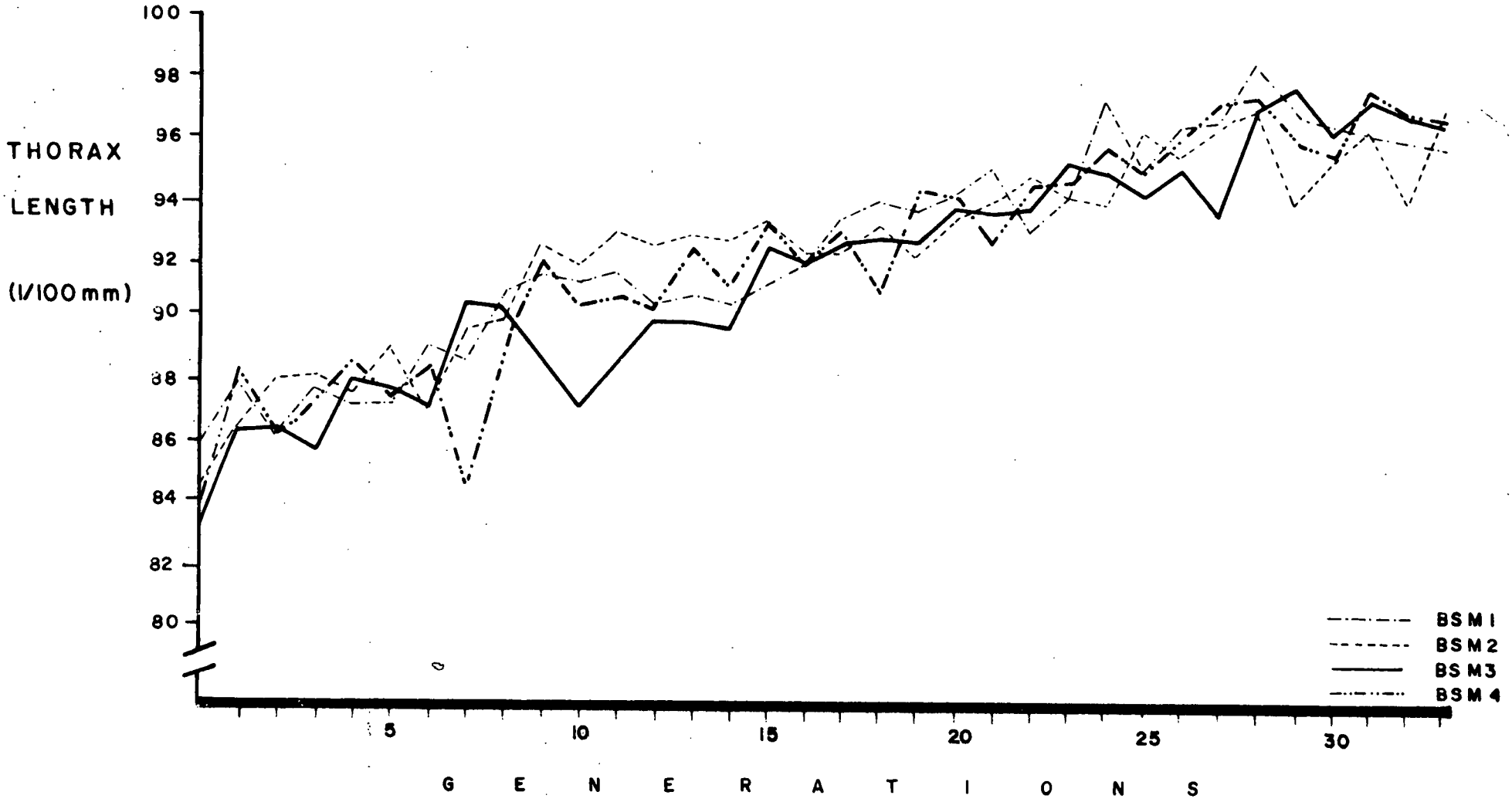


FIG. 7 SELECTION RESPONSES OF BSM BODY LINES

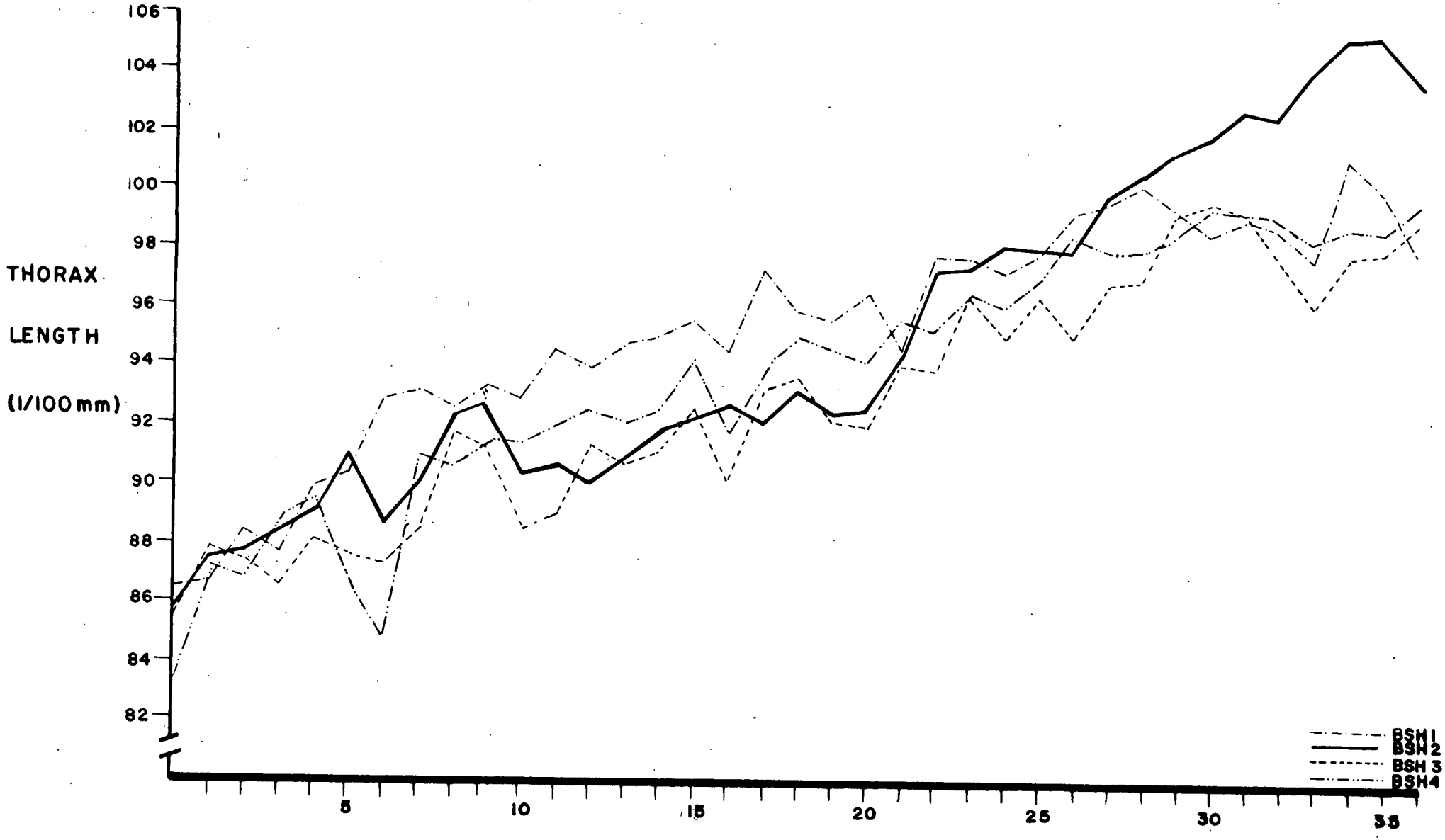


FIG. 8 S E L E C T I O N R E S P O N S E S O F B S H B O D Y L I N E S

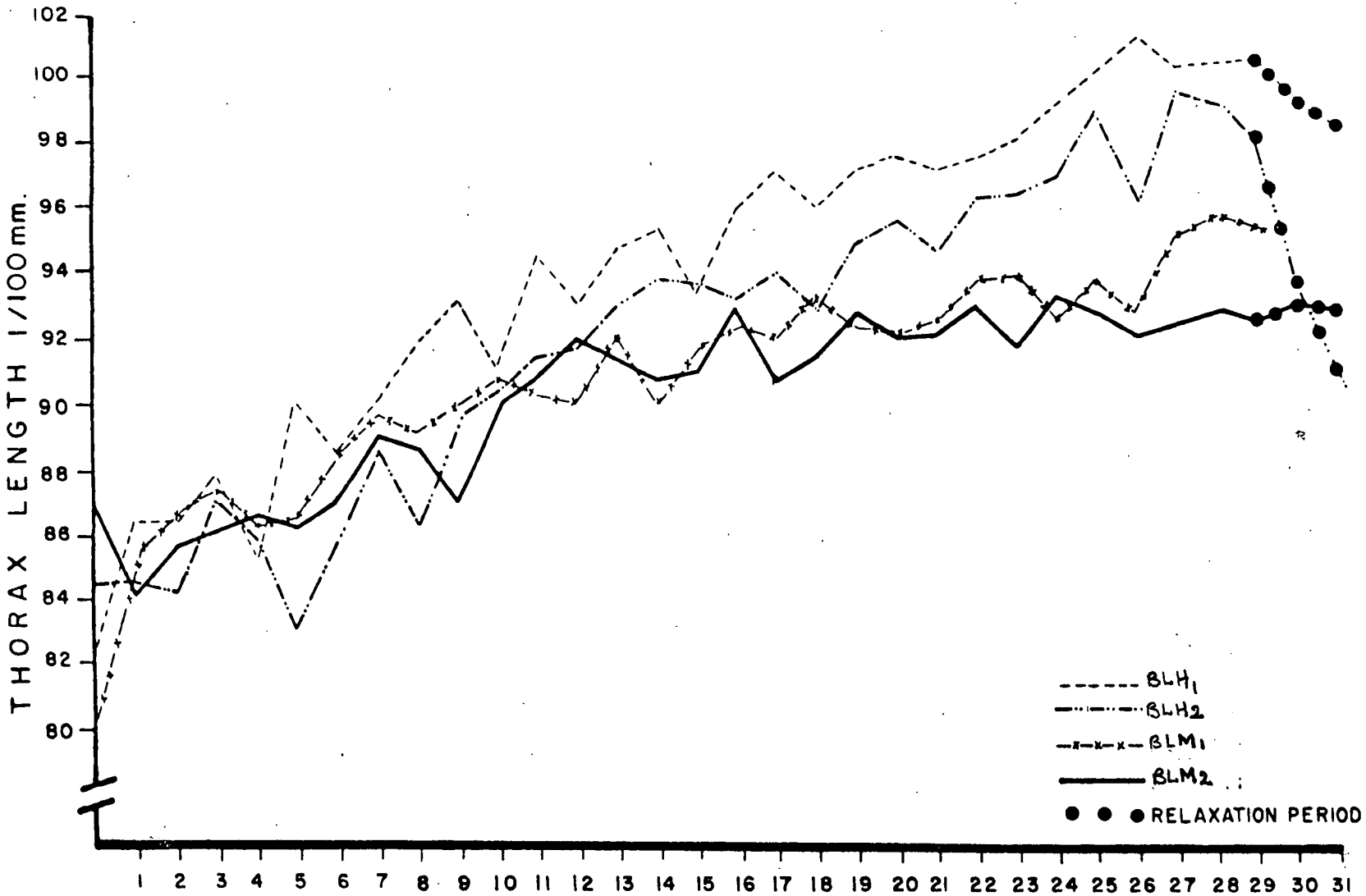


FIG. 9

SELECTION RESPONSES OF BLM AND BLH BODY LINES

TABLE 9. Regressions of response to selection in body length (as deviations from large base population) on generation number.[+]

Generations:	0-10	11-20	20-L
Lines			
$\overline{BSC}$	.31 ± .10**	.02 ± .05	.06 ± .02**
BSM_1	.26 ± .10**	.34 ± .07**	.21 ± .12*
BSM_2	.36 ± .10**	-.05 ± .07	.06 ± .05
BSM_3	.07 ± .21	.52 ± .10**	.29 ± .08**
BSM_4	.13 ± .12	.23 ± .11*	.22 ± .11**
$\overline{BSM}$	.16 ± .06**	.26 ± .06**	.22 ± .07**
BSH_1	.46 ± .17**	.19 ± .09**	.54 ± .38
BSH_2	.28 ± .11*	.26 ± .10**	.57 ± .04**
BSH_3	.32 ± .17*	+.29 ± .13**	.20 ± .07**
BSH_4	.26 ± .17	.28 ± .09**	.14 ± .04**
$\overline{BSH}$	.25 ± .12*	.26 ± .08**	.25 ± .04**
$\overline{BLC}$	.15 ± .10	.14 ± .11	.28 ± .10**
BLM_1	.65 ± .16**	.12 ± .12	.06 ± .18
BLM_2	.20 ± .19	.006± .17	-.23 ± .15
$\overline{BLM}$	.35 ± .13**	.06 ± .14	-.04 ± .18
BLH_1	.36 ± .23	.19 ± .15	.21 ± .14
BLH_2	.69 ± .12**	.31 ± .15*	.20 ± .21
$\overline{BLH}$	.41 ± .15**	.25 ± .13*	.20 ± .17

[+] The values of control lines averages ($\overline{BSC}$ and $\overline{BLC}$) are regressions of changes in mean per generation as deviations from large base population on generation number.

and discussed at length by Robertson (1955). BSH_3 and BSH_4 tended to decrease their rate of response slowly throughout the whole period but at the very end decreased dramatically.

Control lines increased their mean in the early period. $\overline{BLC}$ kept increasing at the same rate up to generation 20 to increase at a higher rate in the last period. This behaviour was mainly due to BLC_1 as BLC_2 after the early period maintained its mean about the same level thereafter. $\overline{BSC}$ had its mean increased in the early period and then it was almost unchanged. However, BSC_2 line had a striking increase from generation 18 onwards.

Half-lives of selection response are presented in Table 10. Average values of small population size lines are closer to Robertson's (1960) expectation than those of large lines. Half-life of a line tells us much about the behaviour of it. Lines which start with great response and then slow down early will have short half lives. This is the case of BSH_1 and BLM_2 (see Figs. 8 and 9, and Table 10). However, if a line starts responding badly and then rises in the last period, it will have a long half life as was the case of the BSH_2 line. If a line keeps its rate of response throughout the period and then slows at the end it will have a long half life. This was shown, by BSM_3 and BSM_4. Small population size lines had different patterns of response among them and this is reflected in the great variability

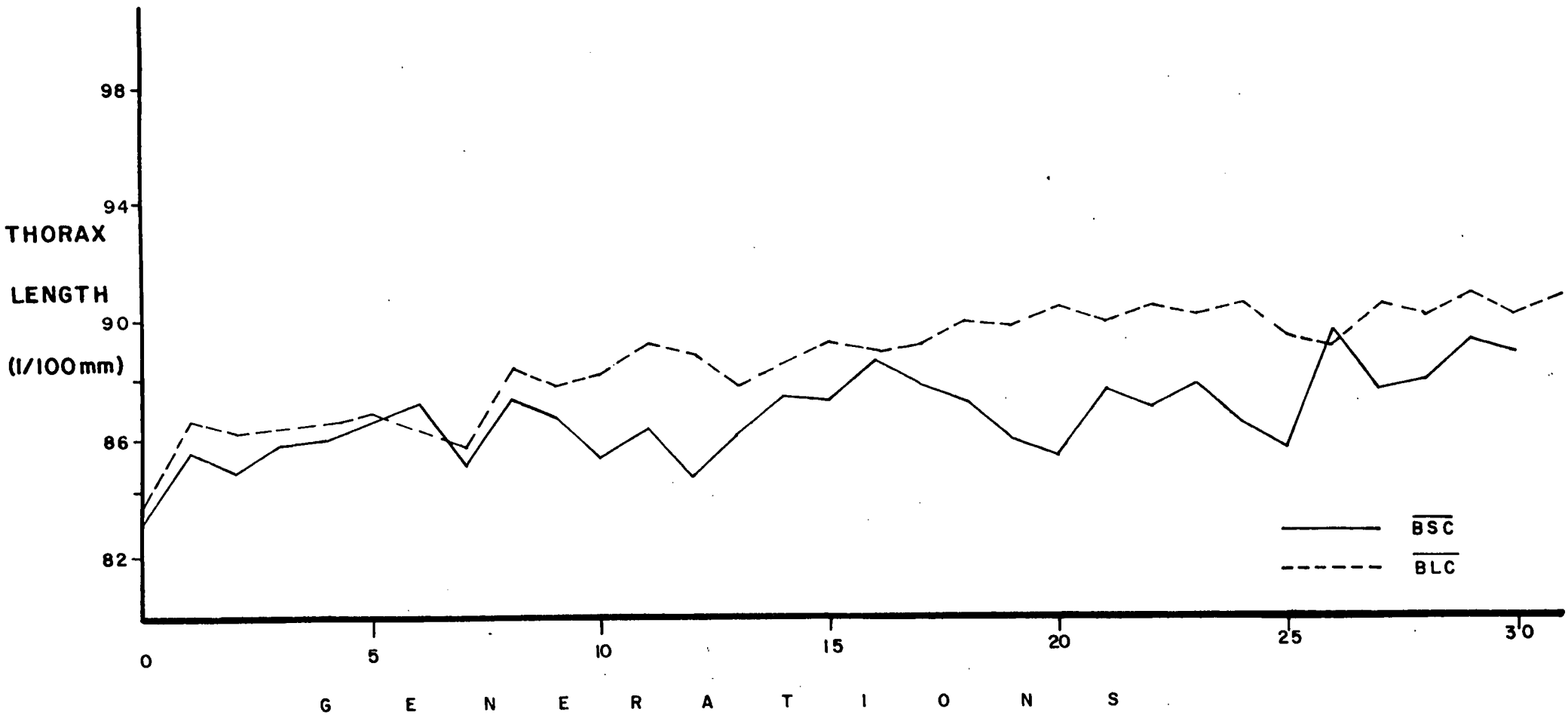


FIG. 10. $\overline{BSC}$ AND $\overline{BLC}$ LINES THORAX LENGTH MEANS

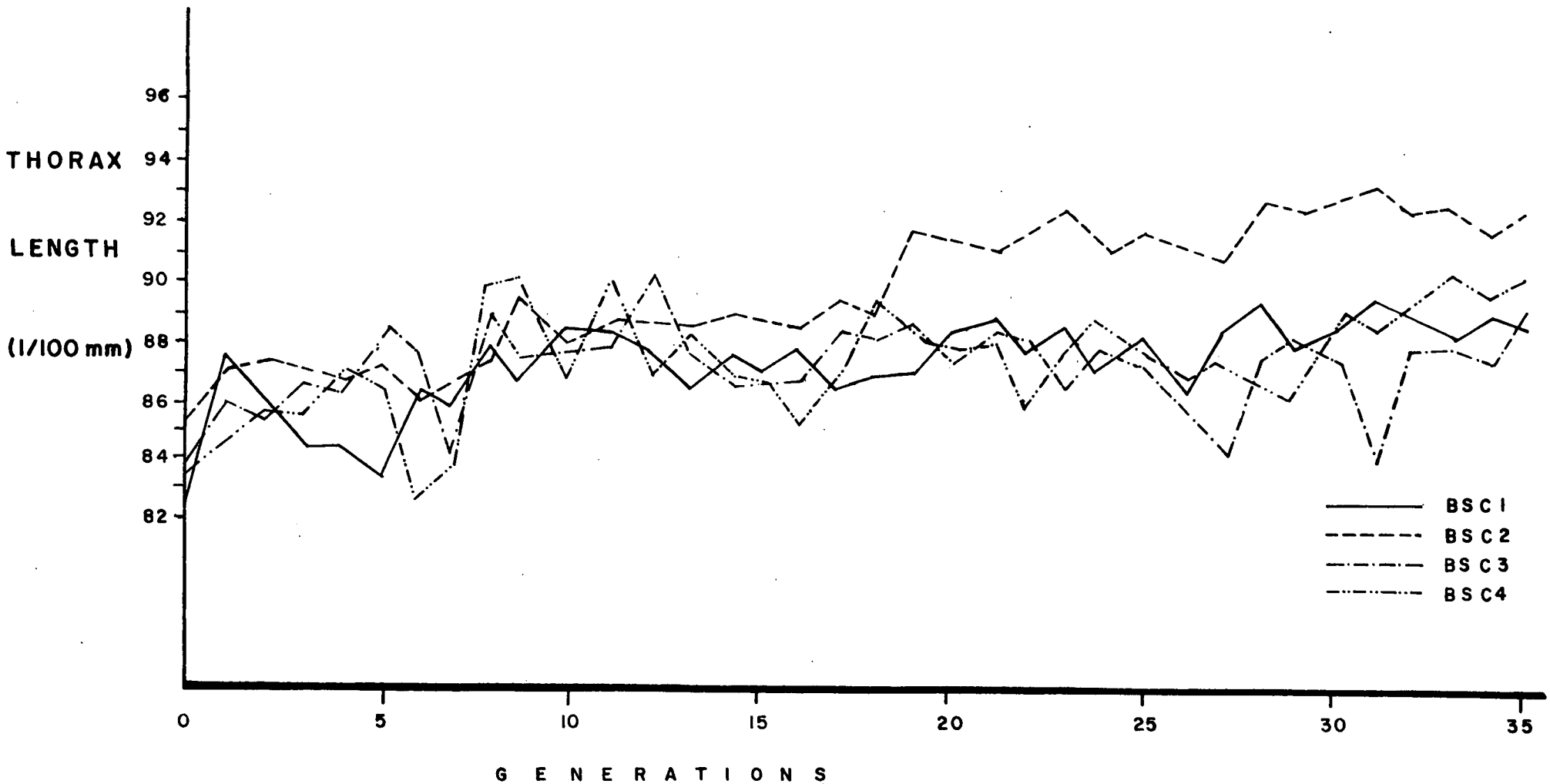


FIG. 11. THORAX LENGTH MEANS OF BSC LINES

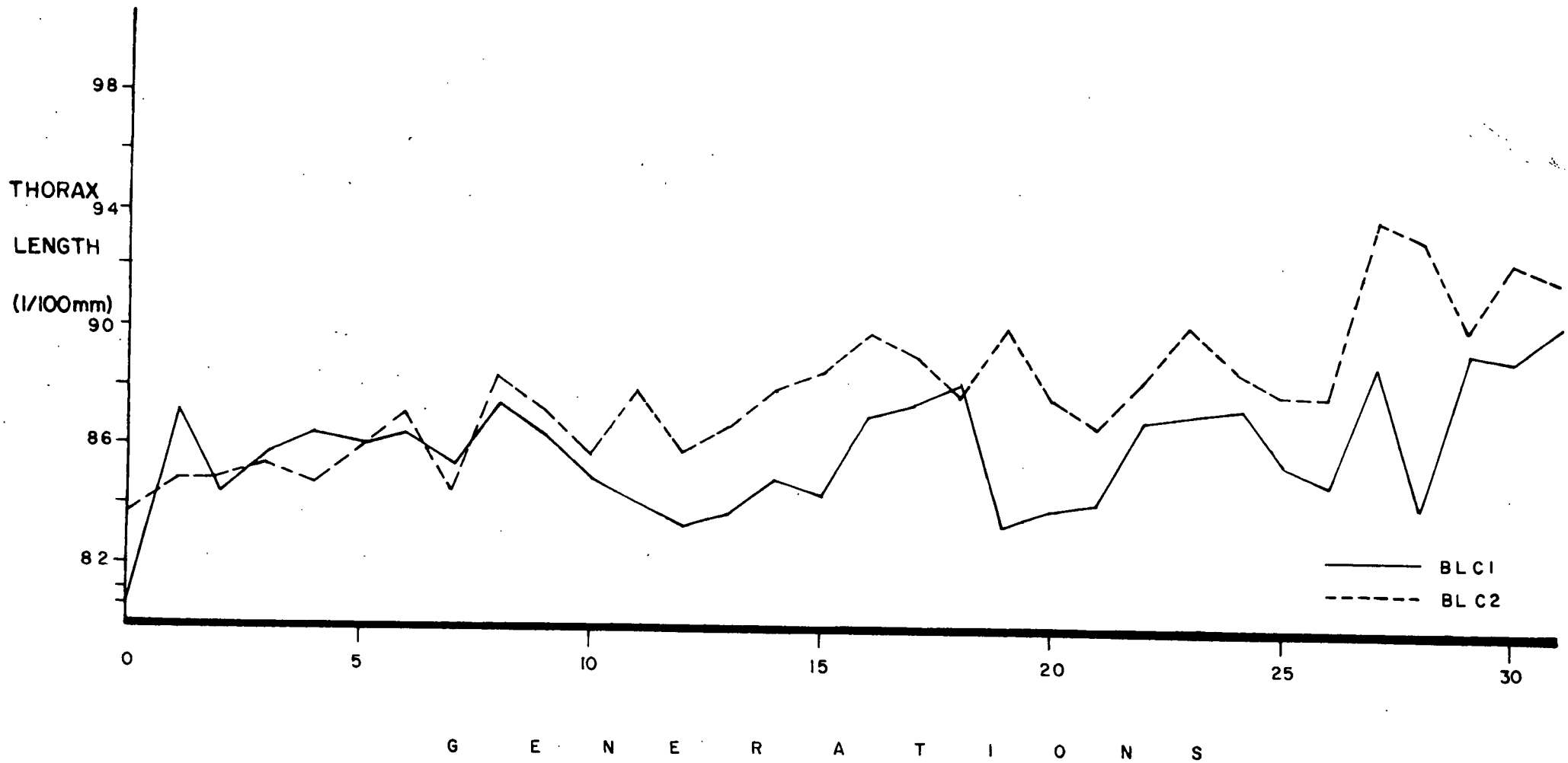


FIG. 12. THORAX LENGTH MEANS OF BLC MEANS

of their half lives. BLM lines had also different patterns of response and only in the intermediate part of the response period were they much the same. It is difficult to use Robertson's (1960) comment on the possibility of having fixed all the favourable alleles if the half-lives are well below the N-2N range in order to explain the low half-life values of BLM and BLH lines. There is still variation in those lines and it is unlikely that all the favourable alleles have been fixed. Perhaps it would be safer to take Roberts' (1966a) position and argue that some other forces rather than fixation are producing the limit.

TABLE 10. Half-life of selection responses.

LINES	GENERATIONS			LINES	GENERATIONS		
$^{+}BSM_1$	9	=	.75N	BSH_1	6	=	.5N
BSM_2	9	=	.75N	BSH_2	22	=	1.8N
BSM_3	15	=	1.26N	BSH_3	16	=	1.34N
BSM_4	14	=	1.09N	BSH_4	13	=	1.09N
$\overline{BSM}$	11.75	=	.98N	$\overline{BSH}$	14.25	=	1.19N
$^{++}BLM_1$	11	=	.22N	BLH_1	11	=	.22N
BLM_2	8	=	.16N	BLH_2	10	=	.20N
$\overline{BLM}$	9.5	=	.19N	$\overline{BLH}$	10.5	=	.21N

$^{+}N = 11.99$ $^{++}N = 48$ using Crow's (1954) correction.

Heritability estimates in generations 5, 10 and at the limit were obtained simultaneously for all the lines. (see Table 11). Although they have large standard errors, I suggest that they were reduced as the selection process went on, but not in some large size population lines. This has been observed by Frankham et al. (1968b) in abdominal bristles of Drosophila, Eisen (1975) in post weaning gain of mice and Robertson (1955) in body length of Drosophila. I would claim, as well, that some lines still have genetic variation, if not all. There are more claims of this sort in the literature than otherwise. Roberts (1966a) considered exhaustion of the additive genetic variance as an explanation of the limits attained in the experiments he discussed. Robertson (1955) also took that view for the limits of their small body size lines. Here, BLM and BLH lines maintained genetic variation throughout the process. The BSH lines increased their heritabilities at intermediate generations and then decreased it at the end, whereas the BSM lines showed a very heterogeneous behaviour.

It is interesting to have an idea of how many loci which affect the character selected for are segregating in that population (n) and what is the mean size of the proportionate effects of them, ($\alpha = a/\sigma$). Although the known procedures give rough estimates of these quantities, that is better than nothing. Using Roberts' (1966) effective population size procedure we got estimates of $\hat{\alpha}$ in the range of .22 to .42 and for n of 20 to 50. The

TABLE 11. Heritability estimates of body length in the 5th, 10th generations and at "the limit".[+]

Lines	h^2		
	5th	10th	L
BSC_1	.191 ± .08	.132 ± .09	-.027 ± .13
BSC_2	.338 ± .07	.073 ± .14	.169 ± .07
BSC_3	.056 ± .13	.097 ± .11	-.103 ± .069
BSC_4	.386 ± .10	.067 ± .08	.060 ± .05
$\overline{BSC}$	.239 ± .046	.091 ± .05	.008 ± .03
BSM_1	.131 ± .13	.164 ± .129	.013 ± .03
BSM_2	.124 ± .09	.064 ± .20	.020 ± .06
BSM_3	.017 ± .18	-.112 ± .21	.310 ± .12
BSM_4	.038 ± .07	.160 ± .18	.297 ± .09
$\overline{BSM}$	.080 ± .05	.020 ± .09	.134 ± .04
BSH_1	-.060 ± .19	.200 ± .13	.285 ± .12
BSH_2	.398 ± .14	.490 ± .20	.024 ± .11
BSH_3	-.119 ± .09	.312 ± .30	.174 ± .21
BSH_4	-.063 ± .13	.202 ± .13	.070 ± .10
$\overline{BSH}$	.012 ± .07	.293 ± .10	.139 ± .06
BLC_1	.038 ± .07	-.017 ± .06	.059 ± .13
BLC_2	.258 ± .07	.192 ± .22	-.119 ± .04
$\overline{BLC}$	.137 ± .05	.060 ± .08	-.084 ± .04
BLM_1	.042 ± .16	.107 ± .16	.141 ± .10
BLM_2	-.037 ± .11	.330 ± .11	
$\overline{BLM}$	-.005 ± .09	.234 ± .11	
BLH_1	.062 ± .041	.027 ± .06	.166 ± .13
BLH_2	.154 ± .08	.440 ± .27	.087 ± .111
$\overline{BLH}$	.089 ± .04	.099 ± .08	.113 ± .08

[+] These estimates were obtained using Hill's(1970) method, in the way explained in the estimation of genetic parameters section, of this Chapter.

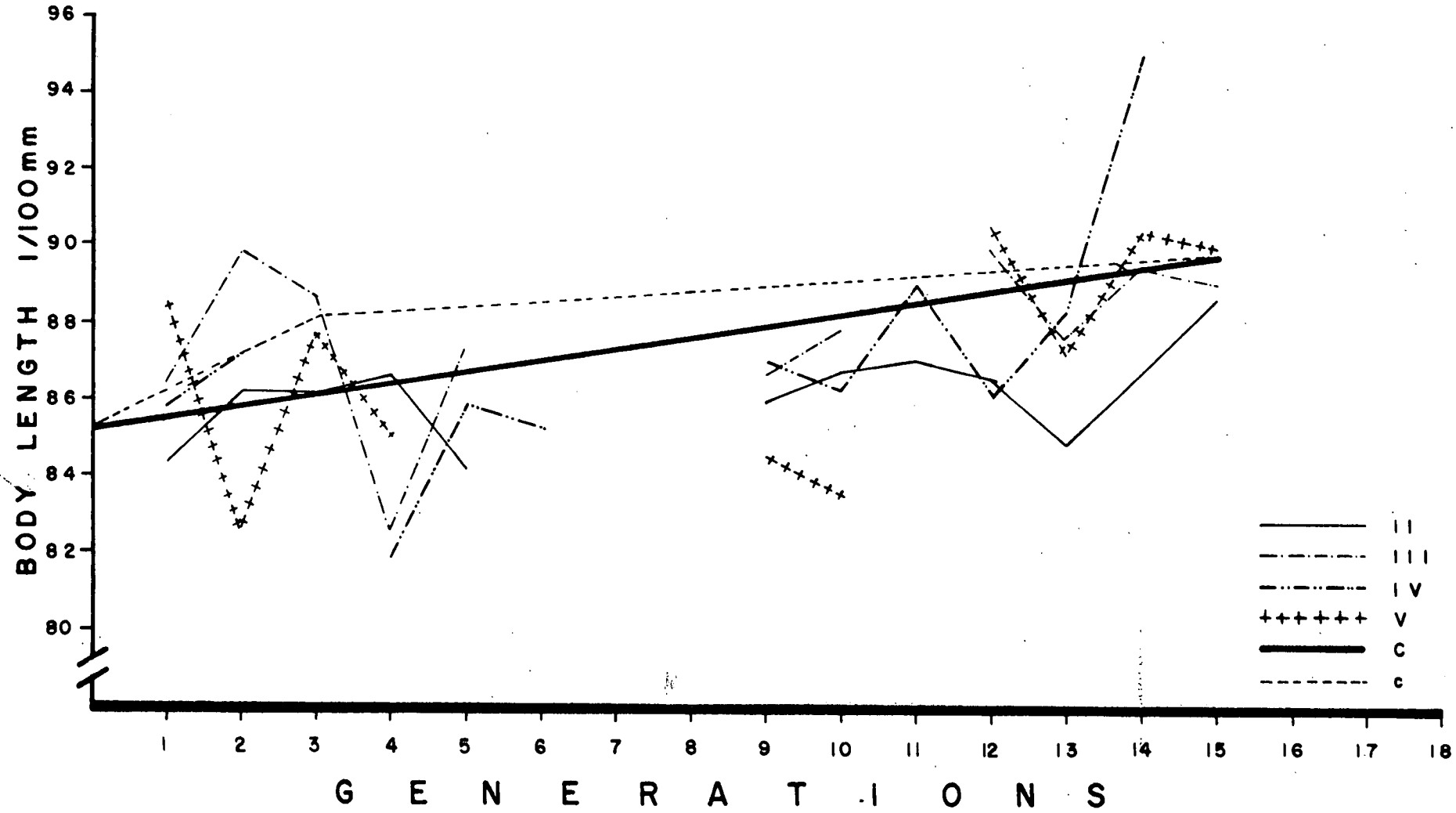
additive approach of Wright (1952) gave us estimates of $\hat{\alpha}$ between .26 to .40 and for n of 7 to 16. The value of $\hat{\alpha}$ most likely as an average proportionate gene effect is close to .25 as three of the four selection treatments gave values close to this figure. The fourth gave a value of 0.42. That would be the best answer if the genetic parameters, we assume are the real ones. Both approaches used gave similar answers. When it comes to the effective number of genes there is a great difference between the effective population size approach and that of Wright. The former gave the largest estimates. If we assume that our initial gene frequencies were high, that leads to consider a value of n about 18 as a good guess for the number of effective genes we are dealing with. It will be the best compromise between both approaches. Reeve and Robertson (1953) considered that 3 or 4 genes with major effects will account for the variation of body length of their lines.

Results of Inbreeding.

In order to know more about the genetic properties of the Dahomey population I was working with, 4 inbred lines were developed using a full-sib mating system for 15 generations. After that, the lines were crossed. Figure 13 presents the changes of the means of the inbred lines over the 15 generations. A continuous thick black line represents the large control population which

FIG. 13.

INBRED LINES
BODY LENGTH



showed an increase in mean of about .133 (1/100mm) per generation. However, as this line was the best empirical fit of 3 points, I feel that the better pattern of change of this large population is that shown by the broken black line. It explains the early rise that all lines had and I think it was caused by a carry over effect, due to the improvement of the environment just before the experiment started. An analysis of variance was performed with the values of the crosses and inbred lines. A comparison of means using contrasts is presented in Table 12.

TABLE 12. Comparisons of inbred lines, crosses and control of body length means using contrasts.

	Inbred Lines			Control	Crosses			
	V	III	II	C	IIxIII	VxII	VxIII	
means	88.1	89.3	89.3	89.8	88.4	90.6	93.1	signif-icance
contrasts								
L_1	-1	-1	-1	3				2.7
L_2	-1	-1					2	8.8*
L_3		-1	-1		2			1.8
L_4	-1	-1				2		3.9*
L_5	-1	-1	-1		1	1	1	5.4*
L_6				-3	1	1	1	2.8

Standard error = 1.23 * .05 Statistical significance level

C.V. = 1.6%

The absence of significant differences between control and inbred lines (L_1 contrast) is confirmed. This is similar to the results of Tantawy (1957) for cousin matings. However, Robertson (1955) and Robertson and Reeve (1955) claimed inbreeding depression in this character. Two crosses showed significant heterosis (L_2 and L_4 contrasts), the average of the crosses show significant heterosis (L_5 contrast) also. This is in agreement with Tantawy's (1957) results. The crossbred lines average was not significantly statistically different to the control although it was larger.

At the end of the selection program, crosses were performed among selected, control and inbred lines. Table 13 gives the mean values of the crosses and Figure 14 summarizes them. There, mid-parent values are on the thick straight line and crossbred lines means are above their respective mid-parent values. The difference is a measurement of the amount of heterosis present in that cross. Crossbred line means are averages of reciprocal crosses. Only the (5) mating did not have a reciprocal. These crosses were planned to enquire further about the nature of gene effects present, and about the inheritance of the character body length. The diallel mating of two inbred lines (Tables 13, set 1) tells us that inbreeding depression was absent and it can not be used to explain any

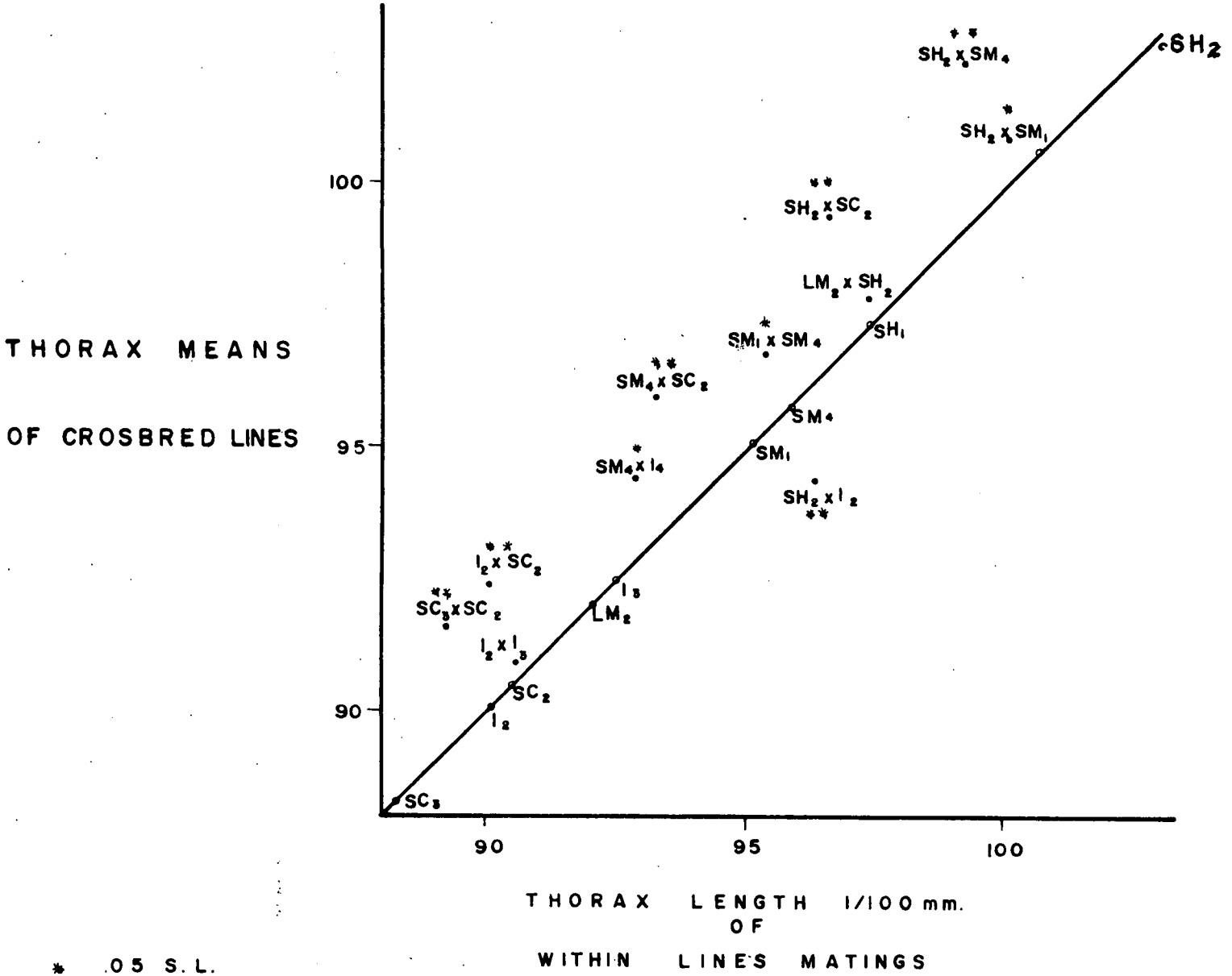


FIG. 14. HETEROSIS OF CROSSES PRESENTED IN TABLE 13.

TABLE 13. Body length means of crosses between selected and control body lines and inbred lines at the end of the selection program.[+]

		H[++]			H
(1)	$I_2 \times I_2$ 90.14±.34 $I_2 \times I_3$ 89.10±.31 $I_3 \times I_2$ 93.42±.36 $I_3 \times I_3$ 92.35±.28	(.02)	(6)	$BSM_1 \times BSM_1$ 95.10±.31 $BSM_1 \times BSM_4$ 97.82±.31 $BSM_4 \times BSM_1$ 95.67±.35	(1.27)*
(2)	$BSC_2 \times BSC_2$ 90.35±.42 $BSC_2 \times I_2$ 93.95±.32 $I_2 \times BSC_2$ 92.32±.28	(2.89)**	(7)	$BSH_2 \times BSH_2$ 102.75±.40 $BSH_2 \times I_2$ 90.28±.54 $I_2 \times BSH_2$ 98.03±.39	(-2.29)**
(3)	$BSC_3 \times BSC_3$ 88.10±.54 $BSC_2 \times BSC_3$ 91.67±.27	(2.47)**	(8)	$BSH_2 \times BSC_2$ 100.67±.31 $BSC_2 \times BSH_2$ 97.75±.25	(2.66)**
(4)	$BSM_4 \times BSM_4$ 95.85±.28 $BSM_4 \times I_2$ 95.04±.39 $I_2 \times BSM_4$ 94.67±.31	(2.26)*	(9)	$BSH_2 \times BSM_4$ 102.64±.26 $BSM_4 \times BSH_2$ 102.20±.36	(3.12)**
(5)	$BSC_2 \times BSM_4$ 95.90±.44	(2.80)**	(10)	$BSH_1 \times BSH_1$ 97.30±.37 $BSH_1 \times BSH_2$ 101.71±.35 $BSH_2 \times BSH_1$ 100.03±.31	(.85)*
			(11)	$BLM_2 \times BLM_2$ 92.01±.37 $BLM_2 \times BSH_2$ 97.28±.28 $BSH_2 \times BLM_2$ 98.10±.39	(.31)

μ = 89.52±.2

[+] 20♂ and 20♀ were measured in each crossbred progeny. Values in 1/100 mm.

μ=Mean of the large base population.

[++] H stands for heterosis of these crosses.

behaviour of the small population size lines. These crosses showed no heterosis either. It seems that the genes affecting variation of body length in our Drosophila population were acting additively. We found a similar situation after 15 generations of brother x sister mating (see Table 12). Tantawy (1957) and Robertson and Reeve (1955a,b) found completely different results. However, Tantawy (1957) with a slower rate of inbreeding found no inbreeding depression for body length although the hybrids showed significant heterosis.

Small control lines (Table 13, sets 2 and 3) did not have a mean body length different from that of the large control, which indicates that the reduction of hetero zygosity due to the drift undergone for those lines did not have any effect on the measurement of the character under study. However, when we cross BSC_2 lines either with I_2 or BSC_3 (Table 13, sets 2 and 3), the crossbred progeny showed heterosis. This is what was found by Tantawy (1957). BSM_4 line yielded heterosis when crossed to an inbred and a small control line (Table 13, sets 4 and 5). Heterosis was at a lower level when BSM_4 was crossed with a replicate (Table 13, set 6). It seems as if genetic differentiation had primarily caused the hybrids superiority. When BSM_4 was crossed to BSH_2 it gave its highest heterosis level (Table 13, set 9). The progeny of crosses were always near the better parent and only in one case one reciprocal was

higher (Table 12, set 6). The highest and lowest level of heterosis was yielded when BSH_2 was a parent in the cross. The former was with BSM_4 and the latter with I_2. This value, however, is quite suspicious as the reciprocal $BSH_2 \times I_2$ had a low mean value and high variance. I can not find a good explanation for it. Lines more differentiated whether by drift (BSC_2) or drift and selection (BSH_2) gave the highest level of heterosis. However, BSC_2 crossed well with replicates of the same regime whereas BSH_2 did not. Genetic differentiation and development of dominance have to be invoked for an explanation of these results.

4. Discussion

Short-term response to selection:

(a) The early selection response was in good agreement with expectations for $\overline{BSM}$ and $\overline{BLM}$ lines. However, $\overline{BSH}$ and $\overline{BLH}$ lines showed less response than expected. This may be due to a genotype-environmental interaction as was observed by Reeve and Robertson (1953) and Robertson, F. (1960a) when food was a limiting factor.

(b) The early response to selection for body length increased as i increased, but the realized heritability reduced as i increased. Clayton *et al.* (1957), Frankham *et al.* (1968) and Hanrahan *et al.* (1973) found the same results.

(c) Realised heritability and selection response in the short-term increased with increasing population size. This effect of N on short-term response to selection was observed by Lewis and Warwick (1953) and Tantawy (1956) when selecting inbred and outbred populations. Hanrahan et al. (1973) found a significant effect of N between populations of 16 pairs and 8 or 4 pairs of parents. However, when N is not too small, as was the case in Frankham et al. (1968) and in this experiment, the effect of N on short-term response to selection was not so clear.

(d) Agreement between replicate lines was poor and it was poorer between replicates of large population size treatments. The latter is not what we expect and disagrees with Frankham's et al. (1968) and Hanrahan's et al. (1973) reports. One replicate of each of those treatments had very low response causing the great variation between replicates. The early fixation of a major gene causing reduction of body length would explain it. Reeve and Robertson (1953) argued that the presence of major genes affecting body length, as well as the presence of lethals which in heterozygote state increase body length would explain it.

Long-term response to selection:

(a) The effect of population size on selection response at"the limit" was not quite clear. It can be argued that the BLH lines were not at "the limit". However,

the BLM and BSM lines had reached a plateau when they were terminated. Jones et al. (1968) and Eisen (1975) found a marked effect of N even although most of their lines had not reached a plateau yet. A possible explanation of this might be that in our small initial sample there were two or three genes of large effect at very low frequencies and some of small effect at high frequencies. This is in agreement with the low additive variance we found at the beginning in relation to that reported by Robertson and Reeve (1953) and Latter and Robertson (1962). There are outcomes observed in this experiment that would argue as well the presence of genes of large effect. Assuming we were in the situation supposed above, the size of N would not have made any difference to the fate of the genes of small effect at high frequencies. Genes of large effect at low frequencies would have been lost at early generations in our lines of low intensity of selection. This would explain the early plateau and small response of BLM lines and some BSM lines.

Linked genes affecting body length would explain, in part, these results. Hill and Robertson (1966) found that linked genes of large and small effect at low frequencies will have the chance of fixation reduced and this is greater as N increases. Robertson (1970) showed that linkage will reduce further the possible advance as N increases for a given value of i and h*.

In this experiment we started with a small sample and this initial reduction of N could produce the loss of genes at low frequency and it will affect more treatments with high Ni value. James (1962) indicated that if natural selection is present Robertson's (1960) expectation for limits will overestimate them and that the fit to expectation will be worse as N increases.

The presence of lethals as was mentioned in discussing short-terms results, would produce the low response and early plateau of BLM lines.

(b) In general, total selection response increased as Ni increased. The results of Jones et al. (1968), Eisen (1975) and our results for response to selection at "the limit" in the smallest populations tended to be 2N times the response in the first generation. As Ni increased we got less than that as Robertson (1960) had warned. Other causes besides chance fixation of favourable alleles due to small population size would reduce that expectation.

(c) Patterns of response in individual replicates were irregular, but generally as i increased the response curve tended to be steeper. This agrees with Qureshi and Kempthorne's (1968) expectations and Hill and Robertson's (1966) expectations.

(d) Half-lives decreased as N increased. As Ni increased the departure from the expected 1.4N of Robertson (1960) increased.

In that paper and in Hill and Robertson's (1966)

it can be seen that for high values of Ni the expected half lives dropped to a fraction of N. Eisen (1974), Roberts (1966) and Jones et al. (1968) reported half-lives in agreement with ours.

(e) Crossing inbred lines did not yield heterosis in body length. However, when they were crossed with small controls or small population selection lines cross-bred progeny showed heterosis. More heterosis was found for body length when lines of different treatments were crossed than when they were replicates of the same treatment. This was found by Robertson and Reeve (1955) as well. It seems as if selection directed the differentiation of lines in someway.

5. Summary

(1) An experiment was carried out to evaluate the effect of N and i on response to selection. Lines of Drosophila melanogaster sampled from the Dahomey population cage, were selected for body length over 30 generations with population sizes of 10 and 40 pairs of parents and selection intensities of 20 and 50% in both sexes, as well as unselected controls.

(2) Short-term responses were in fair agreement with expectations from the estimated base population heritability, but individual replicates showed poor

agreement between them. Genotype-environmental interaction was invoked to explain poor response to selection.

(3) Realized heritabilities reduced as selection intensity increased but absolute response increased.

(4) Selection response and realised heritabilities in the short-term tended to increase as N increased.

(5) The effect of population size on long-term selection response was not consistent.

(6) As Ni increased selection response and realized heritability increased.

(7) Patterns of response of individual lines indicated an early high rate of response, followed by a smooth decline. Lines with low intensity of selection slowed down earlier.

(8) Half-lives tended to increase as i increased and to reduce as N increased.

(9) More heterosis was found when replicates of different treatments were crossed, than when replicates of the same treatment were crossed.

(10) The results were discussed in terms of different theoretical models and using previous experimental results on this issue.

V. SELECTION FOR PUPAE NUMBER

1. Introduction

Reproduction has been a biological function of great interest for geneticists as it is a major component responsible of fitness. It has great importance in selection experiments or programmes as it can limit the amount of selection applied. The total number of eggs laid in the life of a Drosophila female is usually termed fertility. Survival and fertility can be considered as the components which make up fitness (Knight & Robertson, 1957). The number of pupae produced by a fly involves both survival and fertility. In this experiment the actual measurement performed was the number of pupae formed from the first 5 days of egg production and counted 15 days after the parents were introduced into a vial to mate. Gowen and Johnson (1946) found high correlation between total egg production and the number of eggs laid between the 5th and the 10th day of age. This was the period of life in which our flies were laying. The number of pupae counted five days after the flies in a vial start to emerge was highly correlated with total eggs laid and total number of flies emerged. This was checked in trials carried out before the selection programme was set up. As in our flies, development from egg to fly takes 10 days, we expected to count all the pupae coming from eggs laid during the 5 days of oviposition if we

counted pupae the 15th day after the parents were introduced into a vial to mate.

Pupae number as we called this pupae measurement is a trait easy to measure as the larvae climb up out of the food to pupate on the wall of the vials or around the cotton stoppers. It is highly influenced by temperature, food quality and quantity and surface area of larvae feeding.

There was a fair amount of variation for this character in our population (see fig. 15 and Table 14). This can be explained by the fact of its being a combined character of fertility and survival. Martin and Bell (1969) found heritability of .07 and .51 for fecundity and adult emergence respectively. Richardson and Kojima (1965) found realized heritability of .03 and .04 for fertility in within family selection and .16 using recurrent reciprocal selection. This indicates that a large proportion of the genetic variation of fertility is not additive.

Fertility shows inbreeding depression (Gowen and Johnson, 1946; Robertson and Reeve, 1952; Martin and Bell, 1960 and Richardson and Kojima 1965). However, early results of Castle (1906) and Adolph (1920) showed no such effect. In the present work, inbreeding had to reach intermediate levels for it to depress the mean. However, as inbreeding went on the mean number of pupae was depressed severely.

Selection has been carried out for egg production in

Drosophila in very few experiments. Response to selection has been poor in the upward direction, except when methods exploiting non-additive variance were used (Richardson and Kojima, 1965). Selection in the downward direction has yielded fair amount of response. As a correlated response to selection, fertility has been reduced, in spite of the fact that a positive genetic correlation had been estimated in the base population (Martin and Bells 1965, and Robertson F. 1957, Clayton et al 1957).

2. Materials and Methods.

2.1 Genetic material and its handling.

From the data of the first progeny test mentioned in Chapter III the frequency distribution of pupae number was drawn, this is shown in fig. 15. It is not far from normal, with a mean of 75.14 ± 1.5 and a standard deviation of 20.46. Arguing that survival from egg to pupae is high in our population (this was checked in a sample of flies before the selection programme started) we can say that the mean egg production per day of our flies is low, but has a fair amount of phenotypic variability. However, only 10.2% of this is additive as showed by the heritability estimate of Table 14.

Each line was initiated taking a sample of flies from the base population. Each line was kept in individual trays. To initiate a line a number of single pair matings were set up in vials containing fresh food. They

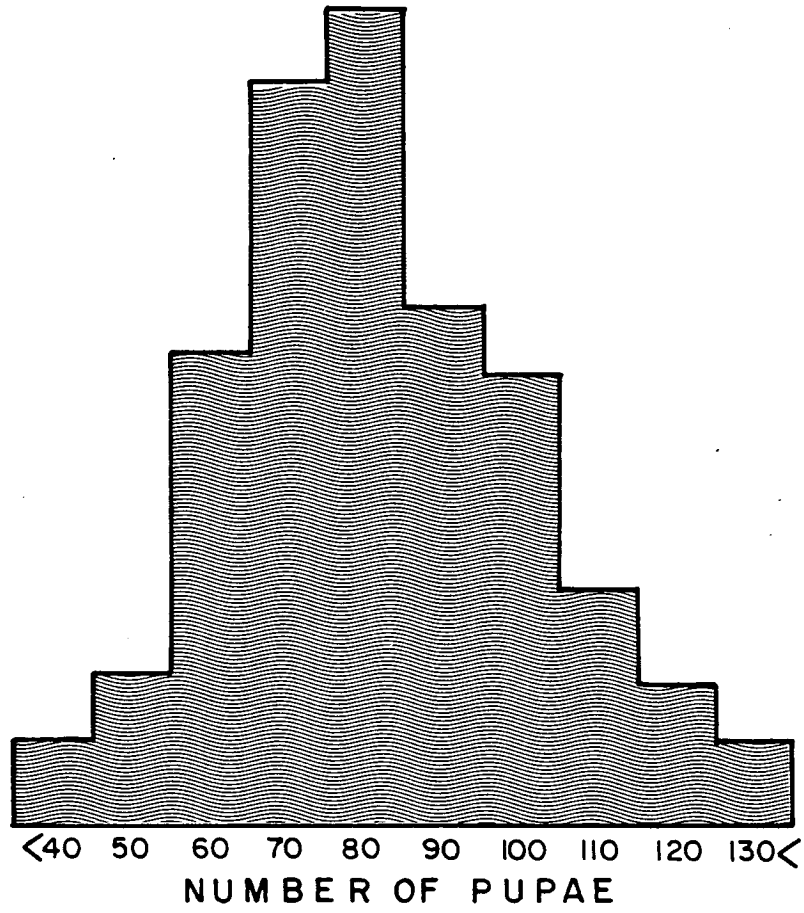


FIG. 15. FREQUENCY DISTRIBUTION OF PUPAE NUMBER

TABLE 14. GENOTYPIC AND PHENOTYPIC PARAMETERS OF PUPAE NUMBER IN THE LARGE BASE POPULATION

n	$\bar{x}$	σ_p^2	h^2	r_p	r_g
200	75,14± 1.5	418.9	.102± .02	-.054± .07	-.958± .10

were shaken out after 5 days. Ten days after this the pupae on the wall of the vial and the cotton stopper were counted. The appropriate number of vials were selected. Females from each selected vial were taken at random, but making sure that each selected vial contributed the same number of females. Single pair matings were set up at random in fresh food containing vials, however, steps were taken to avoid sib-matings. Selection cycles were of 15 days.

It should be noted that selection of a female through the number of pupae counted in a vial includes the fertility of that female and the survival of its progeny up to pupae. The lines were kept at 25^{o}C, except when they were taken out for selection. Care was taken to use vials containing food made the same day that a new cycle of selection was started. To get estimations of correlated responses or genetic parameters, the same number of flies were taken from each vial after having taken the flies needed for the selection programme. Pupae were counted directly and if in any case larvae were still coming up they were not considered. Extra vials were always set up to be sure of getting the required number of vials with pupae. The vials to be counted were randomly chosen.

2.2 Estimation of selection response and its analysis.

Selection response for pupae number will be presented as regression of response taken as deviation from own

controls on generation number. These regressions will cover intervals of ten generations. The first interval will be used to characterize initial response to selection. Using these coefficients of regression as variates an analysis of variance was performed to examine the effects of N and i on the short-term response to selection for pupae number. The second interval covered the period in which all the lines were approaching a plateau.

Regressions of response on selection differentials for 10 generation intervals will be calculated to estimate realized heritabilities in these periods.

Figures showing response to selection will be presented in absolute values accompanied by their own controls. Total response was calculated as the difference between the deviation of final value from initial value of a line and the deviation of final value from initial value of its control.

2.3 Estimation of genetic parameters.

In generations 5, 10 and 30, estimation of heritability of pupae number and phenotypic and genetic correlations of this trait with body length were estimated for each pupae number line. Reeve's (1953) and Hill's (1970) methods were used as described in Chapter III for the base population. In this case, 50 pairs of flies were used and the 10 of each extreme were selected and assortatively mated. Three male and three female offspring were measured for body length and 6 female offspring for pupae number. Correlated response for body length was observed

each five generations. From these measurements realized genetic correlations were calculated.

3. RESULTS

3.1 Short-term response.

Response to selection for pupae number is presented in Table 15. The observed values were higher than the expected ones for intermediate intensity of selection treatment averages.

TABLE 15. Response to selection for pupae number (expressed as regression coefficient of accumulative response on generation number) and expected values.

PSM_1	.715 ± .450*	PSH_1	1.820 ± .300
PSM_2	2.630 ± .450	PSH_2	1.980 ± .30
PSM_3	.172 ± .450	PSH_3	.714 ± .30
PSM_4	2.960 ± .450	PSH_4	1.630 ± .30
$\overline{PSM}$	1.370 ± .230	$\overline{PSH}$	1.560 ± .15
Expected value 0.792			1.37
PLM_1	2.71 ± .22	PLH_1	.226 ± .15
PLM_2	2.12 ± .22	PLH_2	-.021 ± .15
$\overline{PLM}$	2.38 ± .15	$\overline{PLH}$	.109 ± .11
Expected value 0.82			1.44

PSM = lines of small size (S) and medium intensity of selection (M), selected for pupae number (P).

PSH = lines of small size and high intensity of selection (H) selected for pupae number.

PLM = lines of large size (L) and medium intensity of selection, selected for pupae number.

PLH = lines of large size and high intensity of selection, selected for pupae number.

The expected values were calculated using parameters of the large base population. The accumulative response is expressed as deviations from own controls.

*Standard deviations were estimated according to Hill (1971).

$\overline{PSH}$ had an observed value similar to the expected one, but the observed value for $\overline{PLH}$ was much lower than expectation.

The pattern of response of pupae number was similar for all the replicate line averages (see fig. 16, a, b). After an early rise, the means kept more or less their same values up to generation 8 in which they increased again. PSM replicates had quite different patterns of responses to selection. However, the other replicate treatments had similar patterns of response (see fig. 17 a, b, c). In general, lines which did not show the late increase had low average response to selection (see PSM_1, PSM_3, PSH_3 amd PLH_2 in figs. 17, a, b, c and table 15). This can be seen as well in fig. 16, b in which $\overline{PLH}$ did not rise in the last five generations.

Table 16 presents the regression coefficients of generation means on generation number for control and selected lines. It shows that control lines had a tendency downwards (but not PSC_4). It follows from the pattern of an early rise and then a continuous decline which is rather clear in $\overline{PLC}$. (See fig. 16, a, b). $\overline{PLH}$ had the same response pattern and had a negative coefficient as well.

TABLE 16. Regression coefficients of accumulative response on generation number of control and selected lines and averages.

PSC_1	-.292±1.30	PSM_1	-.594±1.25	PSH_1	1.051±1.30
PSC_2	-2.002±1.13	PSM_2	1.848± .64	PSH_2	1.242±1.33
PSC_3	-1.356± .93	PSM_3	-.398±1.60	PSH_3	-.859±1.48
PSC_3	.433± .79	PSM_4	2.198±1.16	PSH_4	1.388±1.01
$\overline{PSC}$	-.802±1.03	$\overline{PSM}$	.763±1.16	$\overline{PSH}$	.705±1.28
PLC_1	-.479±1.26	PLM_1	1.401±1.18	PLH_1	-1.098± .92
PLC_2	-.492±1.26	PLM_2	.926±1.02	PLH_2	-1.354± .80
$\overline{PLC}$	-.485±1.26	$\overline{PLM}$	1.163±1.10	$\overline{PLH}$	-1.226± .86

PSC = (N=20 P=100%) Small size control of lines selected for pupae number.

PLC = (N=80 P=100%) Large size control of lines selected for pupae number.

The analysis of variance presented in Table 17 tells us that there is no effect of population size on selection response as is clear from Fig. 16a, b. Selection intensity, however, was statistically significant as we can see from the figures mentioned above. The N x i interaction was not significant. Mean comparisons shown in Table 17 indicate that there is a significant difference between controls and selected lines (comparison (1)), and between intermediate intensity of selection and high intensity of selection, but in favour of the former (comparison (3)). There was no effect of population size between selected lines (comparison (3)).

The realized heritabilities presented in Table 18 were smaller than expected for high selection intensity treatments

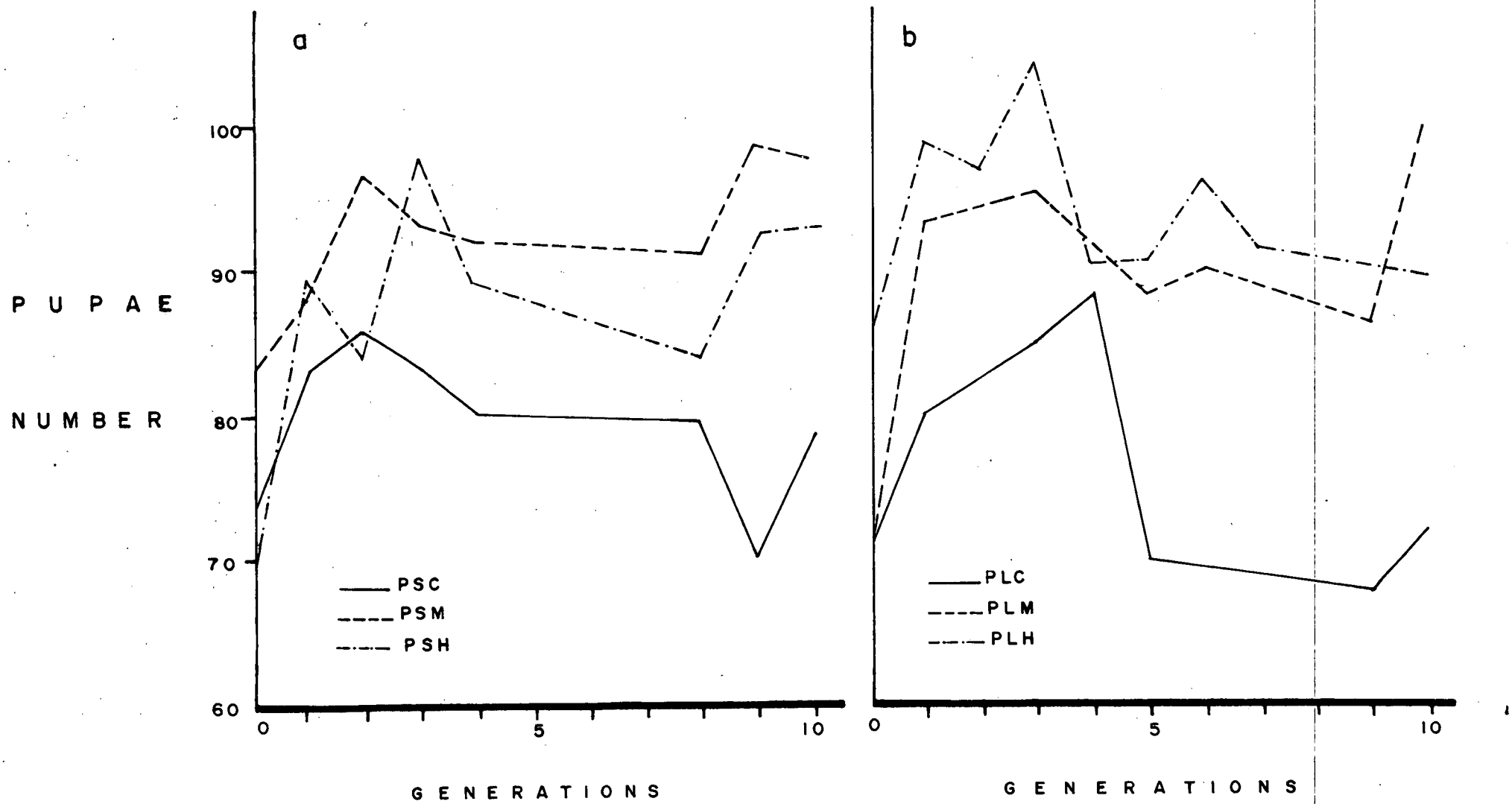


FIG. 16 GENERATION MEANS OF PSC, PSM, PSH, PLC, PLM AND PLH LINES IN THE 10 FIRST GENERATIONS

TABLE 17. Analysis of variance showing the effect of population size and selection intensity on response to selection. The regression coefficients of Table 18 were used as variates.

Source of variation	df	Mean square
Population size (N)	1	.4335
Selection intensity (i)	2	3.8205*
N x i	2	2.4631
Error	12	.8848

Mean comparison (using contrasts)

(1) $2(\overline{PSC} + \overline{PLC}) - (\overline{PSM} + \overline{PSH} + \overline{PLM} + \overline{PLH}) = -3.986^*$

(2) $(PSM + PLM) - (PSH + PLH) = 2.448^*$

(3) $(PSM + PSH) - (PLM + PLH) = 1.529$

* .05 and .01 level of statistical significance, respectively.

($\overline{PSH}$ and $\overline{PLH}$). $\overline{PSM}$ had a $\hat{h}^2$ very similar to the expected one. The lines which responded more (PLM_1 and PLM_2) gave an average with double realized heritability which was expected. This is in line with body length results. Here again it is quite clear and different from expectations that as i increases $\hat{h}^2$ decreases at both levels of N. This was found by Hanrahan et al. (1973) and Frankham et al. (1968) and ourselves for body length. The effect of N on realized heritability depends on the level of i.

TABLE 18. Realized heritabilities for the 10 first generations+

PSM_1	.021±.04+	PSH_1	.077±.02
PSM_2	.246±.06	PSH_2	.116±.02
PSM_3	-.015±.05	PSH_3	.030±.02
PSM_4	.209±.06	PSH_4	.101±.02
$\overline{PSM}$	.107±.02	$\overline{PSH}$	.069±.01
PLM_1	.214±.03	PLH_1	.014±.01
PLM_2	.199±.03	PLH_2	.002±.01
$\overline{PLM}$	.208±.02	$\overline{PLH}$	.008±.007

+ The standard errors were calculated according to Hill (1971).

This leads to an N x i interaction which although existing was not statistically significant (see Table 17 for the analysis of variance of selection response). This outcome is surely due to the poor performance of PLH_1 and PLH_2 lines and mainly to the latter. This has much to do with the effect of larval competition which will be discussed in the next section as it became clearer as selection went on. Notice in Figure 17 that PLH_2 started with a mean of about 95 pupae number and then went downwards all the way through. PLH_1, although starting low, by generation 2 was above 100 pupae number and then decreased and never recovered in the short term.

Inbreeding perhaps did not depress the pupae number

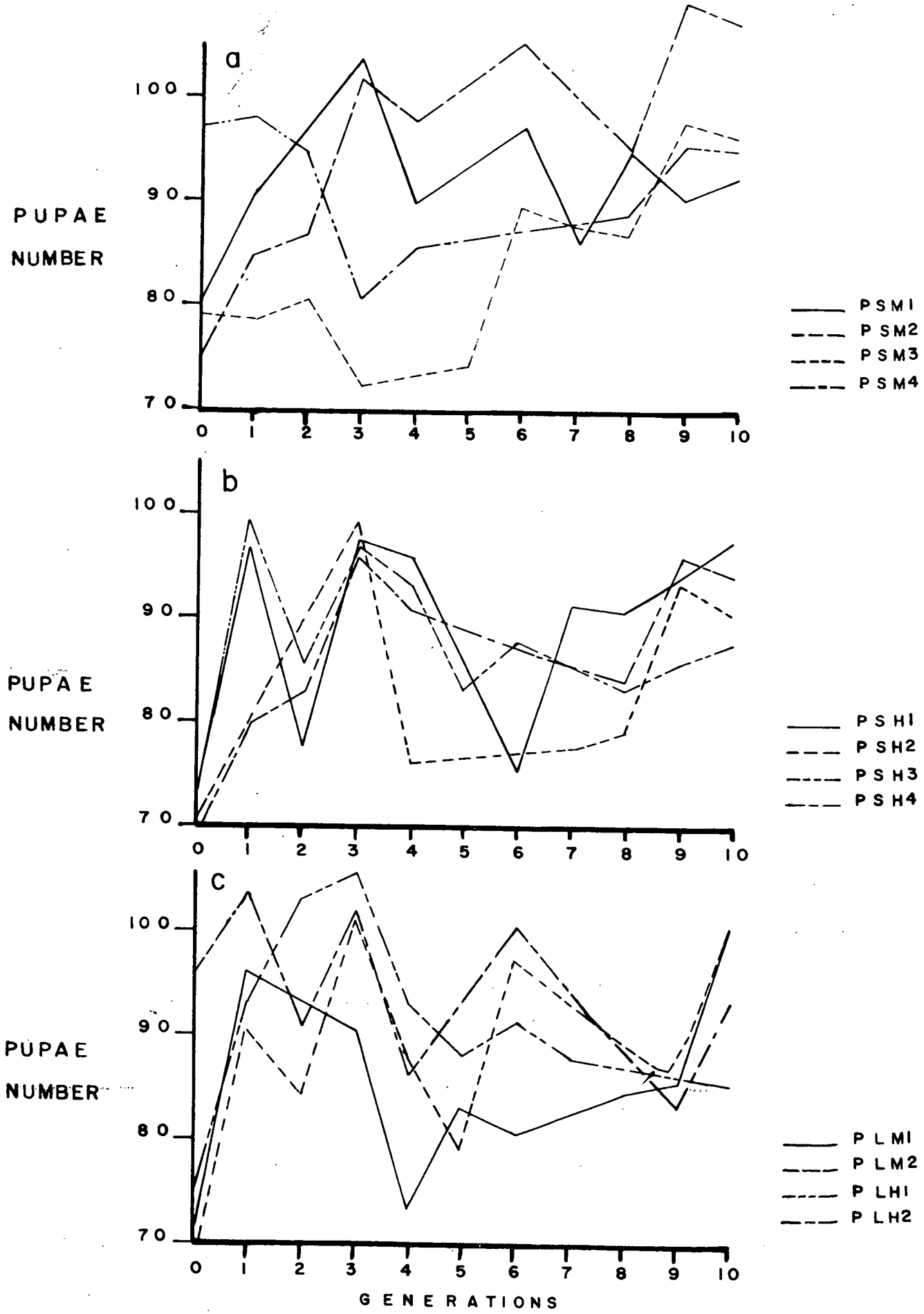


FIG. 17. GENERATION MEANS OF PSM, PSH, PLM AND PLH REPLICATE LINES IN THE 10 FIRST GENERATIONS

mean at generation ten. Our inbred lines started showing inbreeding depression about generation four when the level of inbreeding was theoretically .60. At this point, the mean had been depressed by .5σ, the expected inbreeding coefficient of our small lines without selection is of about .127 which will not be much greater in our selected lines. Martin and Bell (1960) found a decline in egg production of .37 eggs for 1% of inbreeding, therefore we will expect to have a decrease in the mean of about 5 eggs for the level of inbreeding we expected to have in our small lines. Taking into account that larval survival is not much affected by inbreeding and the high variance of the character pupae number, it will be rather difficult to detect inbreeding depression effect if it existed. Therefore on these two pieces of information we can not conclude that inbreeding would have affected our results.

3.2 Long term selection response.

The pupae number trait showed an initial high phenotypic variation ($\sigma = 20.44$). This allowed us to impose large selection differentials. However, as in the case of body length, the phenotypic variance declined rapidly in the first 5 generations in most of the lines (see Figs. 6, 7, 8, 9 and 10 in Appendix). Along with that reduction, less selection pressure was exerted in the selection lines (see Table 3 in Appendix). After the early decline, phenotypic variances remain more or less constant throughout the selection process. This pattern has been found in previous experimental work as

mentioned in the previous chapter.

In those figures a very variable pattern is shown by the lines in the last 10 generations. This is due to the change in the actual measurement. After generation 20, the laying period was progressively reduced to 4, 3 and 2 days as will be explained later. The amount of selection exerted was steadily reduced throughout the selection process being rather clear in PSM lines (see Table 3 in Appendix). The total amount of selection pressure put on the selected lines was of the order 11σ for lines with medium intensity of selection and of 19σ for those with high selection intensity. Considering that selection was only practised on females, it can be seen that more selection was done for pupae number trait than for body length in which 16σ and 30σ selection pressure was accumulated on medium and high selection intensity lines respectively. There was not much variation in selection pressure exerted between replicate lines in PSH and PLM treatments but PSM_1 had much higher selection pressure than its replicates (see Table 3 in Appendix). PLH replicates showed a significant difference as well.

From Figure 17 it is clearly noted that after generation 10 when the selected lines reached a level about 100 pupae number, they did not advance at all in spite of the fact that more selection pressure was exerted on them. This is why it was decided to stop selecting for pupae production over 5 days of egg laying at generation 20. Several experimental trials were performed to give us light on the nature of that ceiling. They will be presented later. Response

to selection then stopped when this ceiling was reached and the difference that we can see in Table 19 between treatments is due to what had happened in early generations.

It is clear from Figure 18a that $\overline{PSM}$ and $\overline{PSH}$ means fluctuated around a pupae number of about 100 from generation 12 to 20, and Figure 18b shows that $\overline{PLM}$ and $\overline{PLH}$ did the same. In the latter figure a differentiation between $\overline{PLM}$ and $\overline{PLH}$ lines is observed, but this occurred before the ceiling of 100 pupae was reached. In Figures 19a, b and c, we can see how the selected line replicates move about a level of 100 pupae number, and several of them reached that peak in very early generations. There is no clear relation of R_1 with either N or i (Table 19). The separate replicates show no clear relation of RL with their values of $\Sigma\delta/\sigma$. What is clear is that something was preventing the selection lines from showing their genetic potential for pupae production.

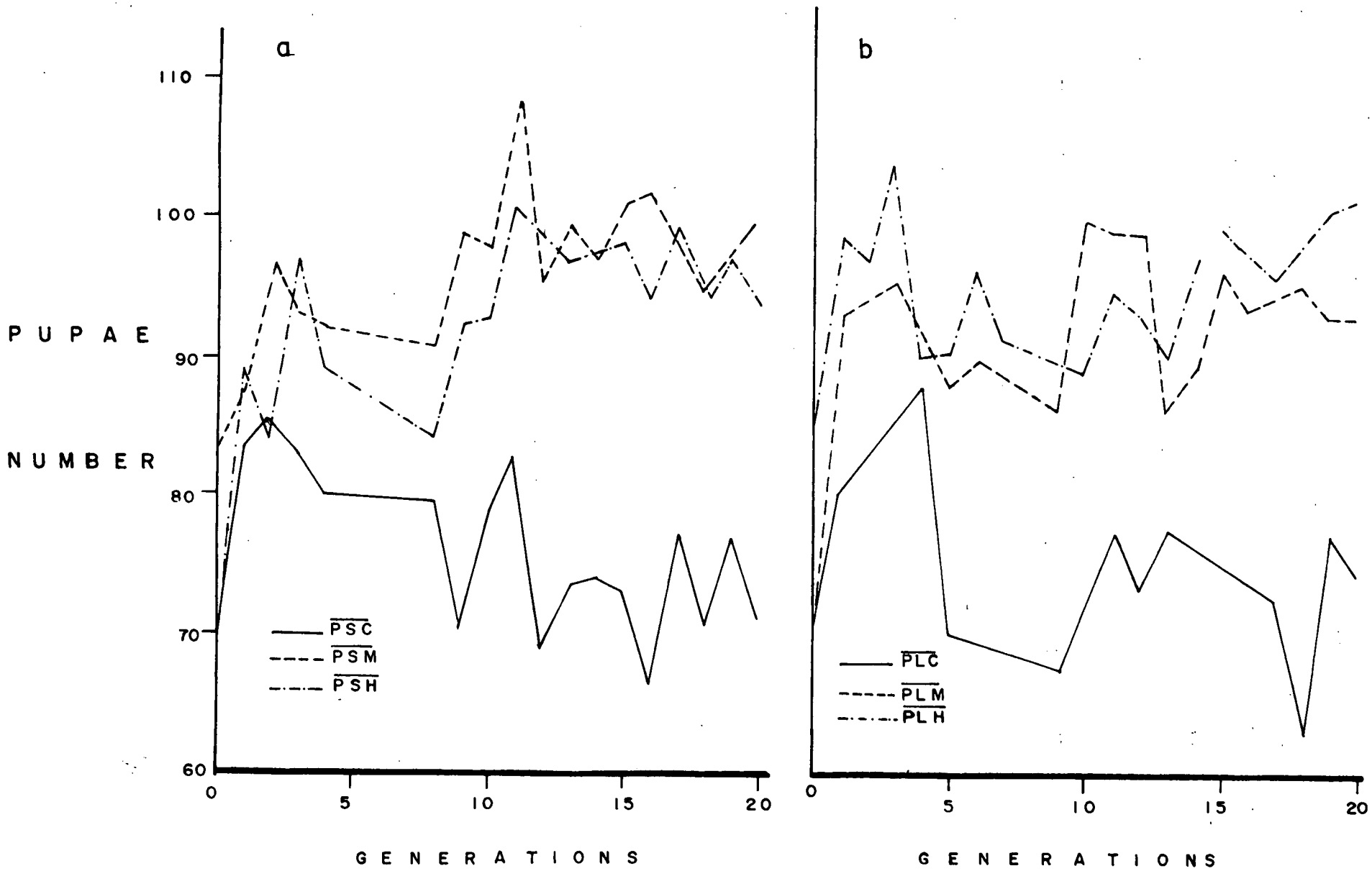


FIG. 18. GENERATION MEANS OF $\overline{PSC}$, $\overline{PSM}$, $\overline{PSH}$, $\overline{PLC}$, $\overline{PLM}$ AND $\overline{PLH}$ LINES

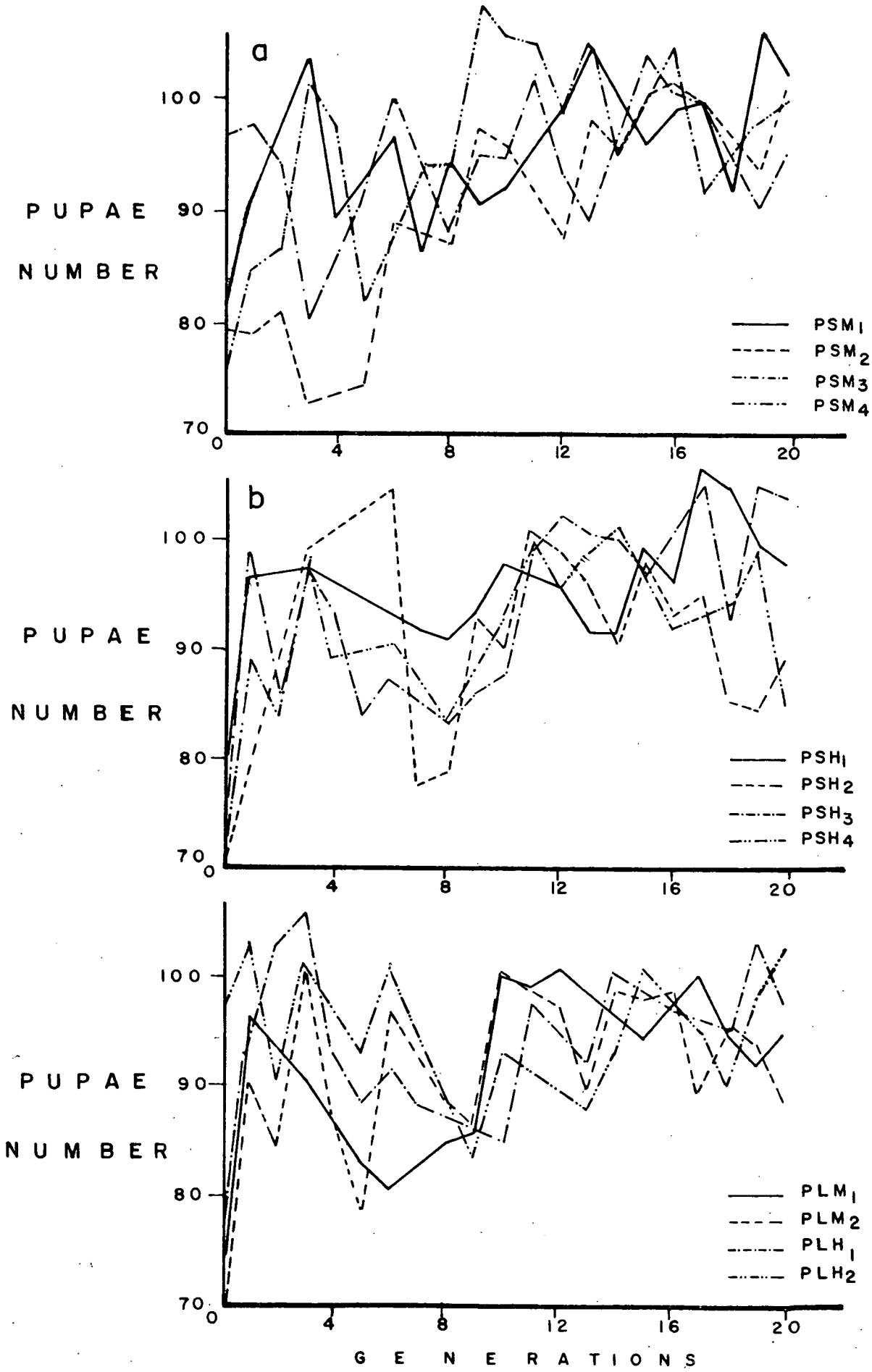


FIG. 19. GENERATION MEANS OF PSM, PSH, PLM AND PLH INDIVIDUAL REPLICATE LINES IN THE 20 FIRST GENERATIONS

TABLE 19. Expected response in the first generation and at "the limit", cumulative selection differential and actual response for pupae number line averages.

Lines	Ni	R_1	RL	$\varepsilon\delta/\sigma_p$	RL_+	RL/σ_p
$\overline{PSM}$	15.2	.792	63.36	11.28	$26.10^{\pm}$	1.2
$\overline{PSH}$	26.4	1.37	109.60	19.06	$23.36^{\pm}$	1.1
$\overline{PLM}$	63.2	.82	262.40	10.41	$23.66^{\pm}$	1.1
$\overline{PLH}$	111.2	1.44	460.80	19.85	$27.14^{\pm}$	1.3

N effective population size, i standardized selection differential, R_1 response in the first generation, RL expected response at "the limit", $\varepsilon\delta$ cumulative selection differential, RL_+ actual response expressed as deviations from own controls.

TABLE 20. Actual response and $\varepsilon\delta/\sigma_p$ of pupae line replicates and averages.

Lines	RL_+	$\varepsilon\delta/\sigma_p$	Lines	RL_+	$\varepsilon\delta/\sigma_p$
PSM_1	$23.26^{\pm}$	13.2	PSH_1	$24.55^{\pm}$	19.6
PSM_2	$26.50^{\pm}$	10.3	PSH_2	$18.15^{\pm}$	18.4
PSM_3	$24.96^{\pm}$	10.8	PSH_3	$27.24^{\pm}$	19.1
PSM_4	$26.10^{\pm}$	9.7	PSH_4	$23.36^{\pm}$	18.1
$\overline{PSM}$	$26.1^{\pm}$	11.2	$\overline{PSH}$	$23.36^{\pm}$	19.0
PLM_1	22.23^{+}	10.5	PLH_1	$27.83^{\pm}$	18.9
PLM_2	$25.09^{\pm}$	10.3	PLH_2	$26.45^{\pm}$	20.9
$\overline{PLM}$	$23.66^{\pm}$	10.4	$\overline{PLH}$	$27.14^{\pm}$	19.8

Heritabilities estimated at generation 5, 10 and 30 (although they have very high standard errors) do not show that there was exhaustion of genetic variability. (See table 21). The several negative values observed may have been due to the environmental ceiling which affected the estimation of them, as will be explained later. For these reasons there was not much point in proceeding before finding out the cause of that ceiling whether it be genetic or environmental.

4. Secondary Experiments

After 15 generations of selection for pupae number it was noticed that there was no further advance in any line and that 11 of them had mean about 100 pupae. As pupae number is a trait with low heritability and with large phenotypic variance it was not a rare event that the line means were varying up and down with little or no increase. Studies by Spiers (1974), on crowding in Drosophila warned us of the possibility of larval competition sometime in our experiment, but we never thought of it at the level of egg production we had at that time. A vial left with a pair of flies for a week or so, yielded about 100 pupae. However, several larvae were still creeping around and then, they died without becoming pupae. It prompted us to set up the following experimental trial.

Experiment 1 - Seven laying periods were put on test with nine replicates per period to see if an increase in the number of days of laying would lead to an increase proportionally in the number of pupae counted. A pair (male

TABLE 21. Heritability estimates of pupae number in pupae lines in the 5, 10 and 30 generations.

Lines	h^2 5	10	30
PSC_1	-.028±.14	-.464±.16	.176±.18
PSC_2	.152±.22	.156±.10	.224±.14
PSC_3	-.302±.14	.380±.16	-.016±.16
PSC_4	.54±.28	.180±.14	.296±.24
$\overline{PSC}$	.092±.10	.058±.06	.172±.14
PSM_1	-.226±.22	.260±.14	.192±.19
PSM_2	.004±.20	-.098±.18	-.464±.20
PSM_3	.356±.22	-.324±.20	-.286±.24
PSM_4	.346±.22	.440±.14	-.092±.21
$\overline{PSM}$	.174±.12	.094±.10	-.162±.21
PSH_1	.084±.30	-.240±.14	.352±.18
PSH_2	.144±.22	-.260±.16	.018±.16
PSH_3	.250±.36	-.042±.14	.050±.22
PSH_4	-.130±.34	-.188±.16	.170±.18
$\overline{PSH}$	.102±.14	.190±.06	.147±.19
PLC_1	.186±.22	.122±.26	.128±.20
PLC_2	.030±.22		-.008±.24
$\overline{PLC}$	.106±.01		.060±.21
PLM_1	.174±.20	.074±.10	.184±.16
PLM_2	.001±.14	-.018±.14	-.016±.14
$\overline{PLM}$	.006±.12	.096±.10	.084±.15
PLH_1	.142±.16	.200±.14	-.132±.20
PLH_2	-.214±.32	.350±.12	.302±.22
$\overline{PLH}$	.044±.14	.276±.12	.085±.21

and female) of flies one day old were introduced in each fresh food containing vial. It can be seen in table 22 that as the egg laying period increased from

TABLE 22. Pupae number counted from different egg laying periods.

Egg laying period (days)	Average number of pupae counted
3	59.3 ± 5.47
4	85.2 ± 7.46
5	100.0 ± 4.18
6	100.4 ± 3.80
7	97.6 ± 4.22
8	106.9 ± 4.33
9	105.6 ± 4.51

3 to 5 days the number of pupae counted increased, but a longer period (6, 7, 8 or 9 days) did not increase the mean. Another thing worthy of notice is that the ceiling point was about 100 pupae.

Experiment 2 - To have more evidence and to widen the range of the laying period, another experimental trial was carried out using the same procedure as before. Four laying periods were used with different numbers of replicates. The results are shown in Table 23.

TABLE 23. Pupae number counted from different egg laying periods. (Second trial).

Egg laying Period (days)	Number of replicates	Average number of pupae counted
3	7	62.85 ± 5.2
6	7	87.40 ± 5.3
9	12	102.40 ± 4.3
12	8	105.62 ± 4.7

Again it was observed that the pupae counted increased as the egg laying period increased, but as soon as the mean reaches 100 pupae, in spite of increasing the days of egg laying the average pupae production remains more or less the same. Even when flies were laying for 12 days the mean pupae counted was 105.62, the curve shown in fig. 20 was empirically fitted to the pupae number. It is clear that as the curve approaches 100 pupae it starts becoming flat. A quadratic curve fitted to the actual values showed a negative coefficient for the quadratic term. This result confirmed those of the first trial. It appears that for some reason about 100 pupae is the maximum we can obtain under our vial conditions. This was possibly causing the observation that our selected lines were not improving in spite of the selection pressure exerted on them.

Experiment 3 - In order to be sure that there was not a problem of egg production that was causing the depression of pupae production we then set up a trial to assess the effect of number of eggs per vial on the viability of

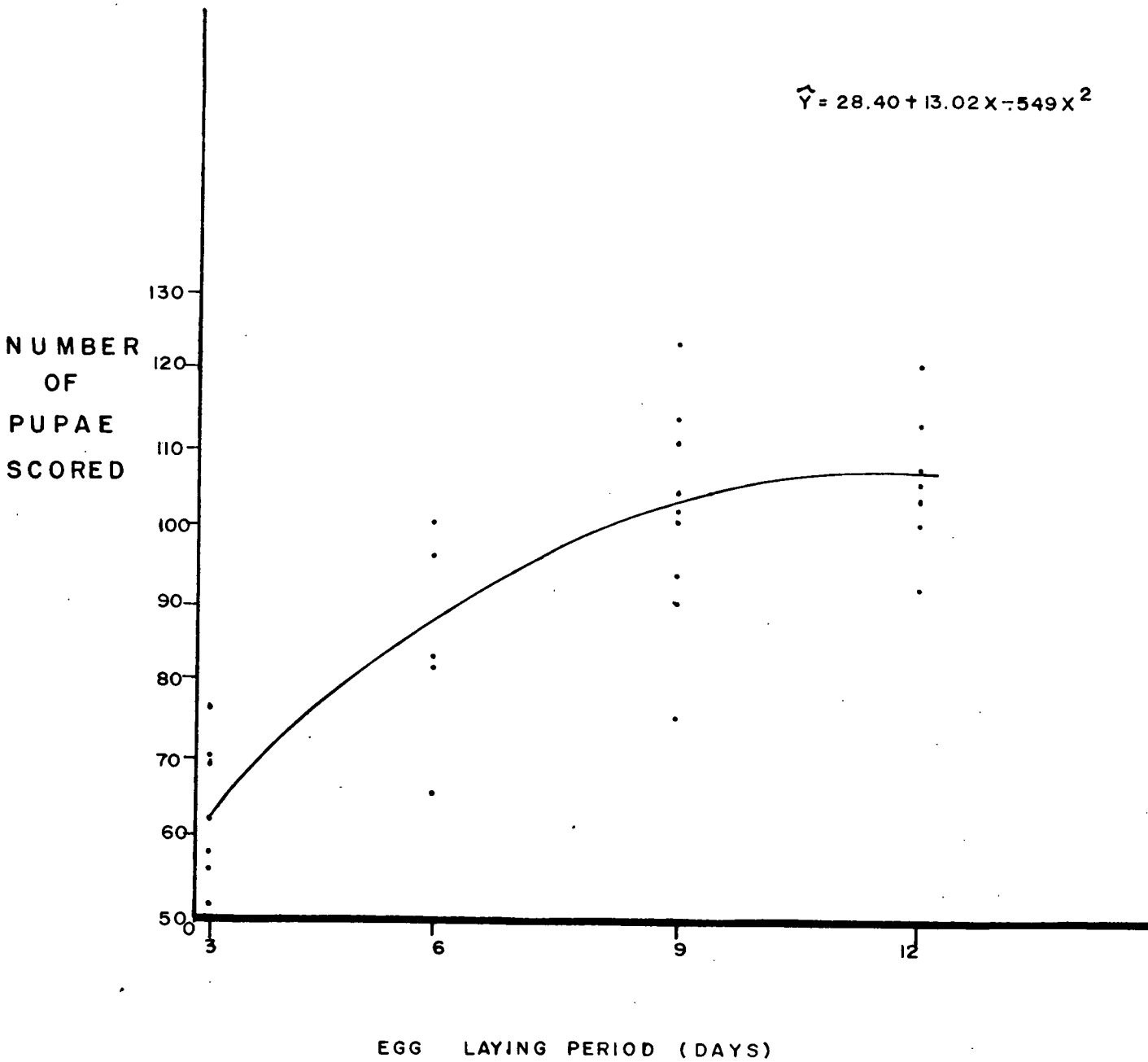


FIG. 20. RELATIONSHIP BETWEEN LAYING EGG PERIOD (DAYS) AND THE NUMBER OF PUPAE SCORED

TABLE 24. The effect of number of eggs per vial on the viability of drosophila flies, from egg to pupa .

Number of eggs per vial	Average number of pupae counted
80	74.6
90	82.6
100	73.6
110	91.3
120	96.6
130	92.3
150	109.0

Drosophila flies, from egg to pupa. We can see from Table 24 that as the number of eggs increases up to 100 there is an increase in pupae number counted, but after that, no matter the number of eggs transferred into a vial, the number of pupae counted is about 100. We then concluded that our vial conditions were not allowing a pupae production much higher than 100 and that the larvae were being affected in their development. Therefore, if by selecting for pupae number we were increasing the egg production capacity of our flies, they were not going to show an increase in pupae production as our vial conditions would have prevented it.

Experiment 4 - Considering that a larva needs to eat certain amount of food to reach a given weight and then become a pupa(Bakker,1961; Robertson, 1963; Church and Robertson 1966; Burnet *et al* 1977) and that larval cannibalism

may exist; an experimental trial was carried out to inquire further on this problem. We thought of giving more food per larva, as well as more room to move around. Three treatments were designed to accomplish this. In treatment A a female was kept in a vial for 5 days. In treatment B a female was kept 4 days in a vial and then transferred to another vial for one more day of egg laying. In treatment C a female was kept 3 days in a vial and then transferred to another for two more days of egg laying.

TABLE 25. Effect of amount of food and space on number of pupae production.

Treatment	Average number of pupae counted
A	78.15 ± 3.1a
B	103.40 ± 3.1b
C	112.00 ± 3.1c

Different letters mean significance of treatment mean differences. 40 replicates per treatment were used.

As we can see, we were counting pupae production over a (5 day) egg laying period; but with different egg crowding conditions per treatment. It is expected that in treatment C larvae had more food per individual and more room to move. Table 25 shows the results of this experimental trial.

As all the flies used were fullsibs, we may conclude that although a fly posseses a certain capacity to produce pupae this will be reduced if there is not enough food, room or both for its larvae to become pupae.

Although there was a great difference between treatments A and B, treatment B mean is not greater than we had achieved in our selected lines. However, it led us to start selecting our pupae lines for pupae production from 4 days of egg laying. After two generations of selection we reached a level of about 100 pupae and after that selection was for pupae production from 3 days of egg laying. In the mean time, we continued investigating the effect of crowding on larval development.

Experiment 5 - Previous experimental trials showed us that in spite of the fact that we increased the feeding surface per larva by transferring females from one vial after 3 days of egg laying to another, there was the possibility that the surface was not sufficient, or that the nutritive value of the food was not good enough to maintain such a number of larvae. Our experience was that larvae do not burrow very deep into the food. The zone searched for food in our vials was only about 1 cm deep. Therefore, there was no point in increasing the amount of food contained in a vial as the feeding volume was not going to increase.

In order to inquire into the effect of feeding surface and food quality on larval development an experiment was carried out. This involved two levels of the factors above mentioned arranged in a 2 x 2 factorial. Each treatment had 3 replicates. Food quality factor levels were (N) normal food and (N + Y) normal food supplemented with 5.3 mg. of live yeast. Feeding surface levels were (V) normal vials and (D) petri dishes. In both containers, the same amount of food was poured. In petri dishes it was spread out to have more

feeding surface. Two hundred eggs laid in a 6 hour period before the trial started were transferred to each container. Fifteen days afterwards pupae were counted. The petri dishes were covered with plastic caps which had a hole at the top, stopped with a cotton ball. The analysis of variance of pupae number indicated that the effect of feeding surface was significant but the effect of food quality and the interaction were not significant. A mean comparison presented in Table 26, showed that it

TABLE 26. The effect of feeding surface and food quality on the development of Drosophila larvae.

Treatments	Pupae number	Treatment means
VN	123	b*
V (N + Y)	117	b
DN	147	ab
D (N + Y)	163	a

* Similar letters for treatments indicates no mean difference. C.V. = 19.5%.

is not the amount of food or nutrients in a vial what is the limiting factor but that the amount of nutrients available to the larvae is the limiting factor.

We could not add more yeast to the normal food of this laboratory without affecting its consistency and texture. As using dishes would have represented a large amount of extra labour, we decided to keep selecting for pupae production from

3 days of egg laying. We carried on for five generations and as we did not see any response, selection was carried out for four more generations using a 2 day egg laying period. As we failed to get any improvement the selection programme was stopped. Fig. 21 shows this late period of the programme. It is clear that no increase at all was obtained. At this stage inbreeding might be affecting response to selection. The big drop in pupae number between a 3 days period and a 2 days period is that in the latter we are missing one day in which egg production is at its highest.

Experiment 6 - Competition for food may reduce the average body length (Robertson 1960a, Sang 1956, Spiers 1977, Burnet et al 1977) as pupae that emerge get only the necessary food to reach the larval weight required to pass to the next developmental state. It is reasonable to assume that the effect of competition on body length will depend on the minimum larval body weight required to pupate, and the first critical growth (Burnet et al 1977). To inquire about this competition effect,as it has relevance to pupae production of selected flies,a test under larval competition was carried out. Based on previous experiments a competition gradient was established by transferring into fresh food containing vials 80, 90, 100, 110, 120, 130 and 150 eggs taken at random from a sample of eggs laid by fullsib flies. Two lines of selected flies were used, one which was expected to have progeny with average body length and the other with large body length. From Table 27, it can be seen that as the number of eggs in a vial increases, there is a reduction of body length up to

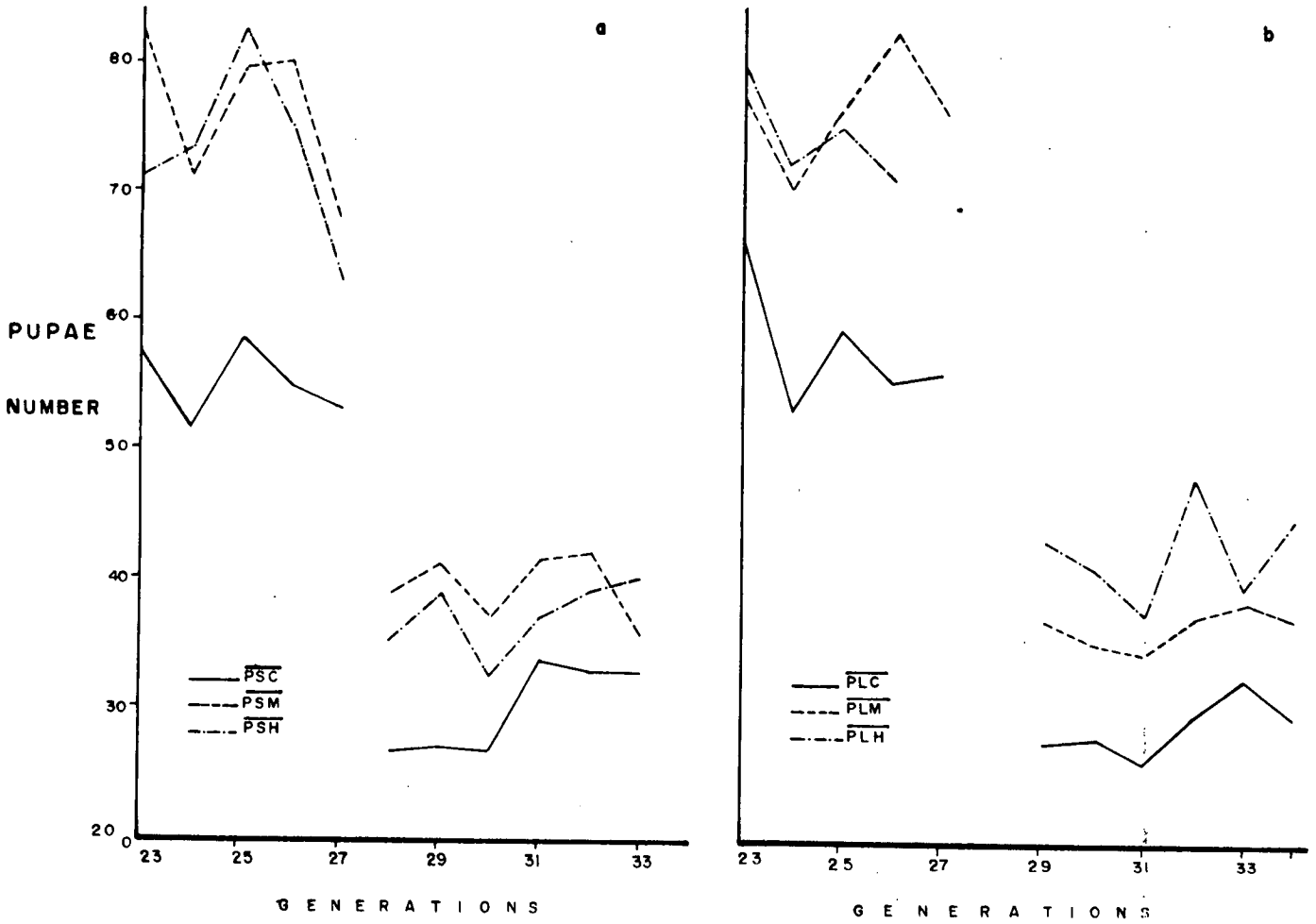


FIG. 21. GENERATION MEANS OF $\overline{PSC}$, $\overline{PSM}$, $\overline{PSH}$, $\overline{PLC}$, $\overline{PLM}$, AND $\overline{PLH}$ LINES BETWEEN GENERATIONS 23 TO 33

a point which seems to be around 100 eggs. Body length of flies of expectedly large size reduced markedly as the number of eggs increased from 80-120. This was not so in average body length flies. These results confirmed our

TABLE 27 Effect of larvae competition on the adult fly's body length

Number of eggs per vial	Body size average (1/100 mm) +	
	Average size	Large size.
80	94.0±.50	97.6±.11
90	93.2±.32	96.6±.12
100	91.6±.10	94.7±.46
110	92.6 .38	95.5±.40
120	93.7±.38	90.8±.46
130	91.6±.21	91.1±.31
150		90.0±.47

+ Only females were considered.

assumption that the larger the potential body length of the adult fly the more severe will be the effect of competition on body length. It has been shown (Sewell et al 1975) that selection can increase the larval feeding speed, but not food efficiency, nor does it affect the critical weight. Therefore, when larvae are under competitive conditions with others having the same minimum weight to pupate, as would be the case among members of a line, larvae which eat faster will have more probability of becoming pupae. However, if food is very limited, each fly will not have much opportunity to get more weight than the critical weight, then its body length will consequently be smaller. The females will lay

less eggs than if their bodies had been larger.

Experiment 7 - Time to pupation is important in our selection experiment as we selected flies at a fixed interval each generation. Under normal conditions larval period is minimized but under sub-optimal conditions larval period can be prolonged (Sang 1956, Robertson 1960a, Burnet et al 1977). Therefore, if competition delayed time of pupation perhaps less pupae would have been counted. To have some insights in this matter, we designed the following trial. First of all, we wanted to compare individuals under competition against individuals with less severe competitive conditions or none at all. Secondly this competition should be similar to our selection programme conditions or nearly so. In other words, competition should increase as more eggs are laid by a fly in subsequent egg laying days. To achieve that and be able to differentiate between eggs laid in different days we used eggs of differant strains each day. The first day we transferred into a fresh food-containing vial 30 eggs of flies of a body length selected line, next day 30 eggs from an Ebony strain were used. Then, the third day 30 eggs of a white eyes strain were put into the vial. The fourth day eggs of the body length selected line were used and the fifth day eggs of the white eye line were transferred into the vial. As we can see, each vial contained at the fifth day 150 eggs of three strains, but ordered in such a manner to avoid confusion at emergence.

TABLE 28 Effect of larvae competition on development time and survival.

*Genotypes ordered	Average time to fly emergence (hours)	(log hrs)	Number of pupae out of 30 eggs	Range of time between the emergence of the first and the last fly of a batch.
B	258±1.68	2.410	28±2.0	1.76±.28
E	277±7.2	2.442	24±2.1	3.72±.51
W	282±5.7	2.451	30±0.0	4.10±.20
B	300±6.9	2.478	26±2.0	4.75±.40
W	305±6.9	2.485	11±2.8	2.44±.37

*B stands for body length. They were eggs from flies of a line selected for body length.
E stands for Ebony. They were eggs from flies of the Ebony stock.
W stands for white eyes. They were eggs from flies of the white eyes stock.

Table 28 indicates that larval competition reduces survival up to a certain level of competition intensity. Above 120 eggs survival was severely reduced in this test. This agrees with all our previous experimental trials. Time to emergence is certainly increased by competition. Average time to fly emergence was calculated from the day the eggs were transferred into a vial to the day of fly emergence. As we used to count our pupae 15 days after a vial had been set up; this factor can be ruled out as causing any reduction in the number of pupae counted.

The range of time between the emergence of the first and the last fly was severely increased as the number of

larvae increased in a vial. It can be noticed that in the last batch of eggs this range was reduced. This is due to the fact that only 11 flies were able to emerge and their emergence was not so spread in time. But if more pupae had been able to emerge, surely it would have taken a longer period between the first and the last one. From these experiments we can conclude that:

a) The ceiling observed in the selection programme of about 100 pupae, was due to the lack of enough nutrients available to keep a larger population of larvae.

b) As egg production was increased by selection, crowding was more severe, then the amount of nutrients per larva was reduced. As a consequence of that survival was reduced, as was the body length of the flies.

c) The average time to emergence was increased by larvae competition but it did not affect the number of pupae we counted for having a fixed generation interval, as we counted at a 15 days interval and the longest time to emergence was of about 13 days.

Inbreeding results:-

Pupae production was reduced by inbreeding. When inbred lines were crossed heterosis appeared and the crossbreds recovered the pupae production lost due to inbreeding (see Table 29).

TABLE 29 Pupae number mean of inbred lines, large base population and crosses of inbred lines.

Matings	Pupae number mean	Average of mating types
$I_2 \times I_2$	52.5	
$I_3 \times I_3$	75.36	
$I_5 \times I_5$	51.70	59.85 [a]
Control	86.78	86.78 [b]
$I_2 \times I_3$	83.88	
$I_2 \times I_5$	83.03	
$I_3 \times I_5$	80.04	82.31 [b]

different letters mean significant difference $P<.05$;
the theoretical level of inbreeding was of .6

Crossbreeding results:-

In later generations we tested several lines using the treatment of transferring a fly to a second vial to lay eggs for the 2 last days of the five days period, but we found pupae productions below 100 pupae. We considered that inbreeding might have been affecting pupae production and so a crossbreeding trial was carried out. Table 30 showed us that inbreeding had definitely reduced pupae number mean as $I_2 \times I_2$ and $I_3 \times I_3$ matings gave values much lower than the large base population mean. All of the small population size lines whether selected or not gave pupae number mean values about 60 when they were crossed. The exception to this was $PSC_3 \times PSC_3$, which yielded a mean of 87.06. Large population size lines gave mean values of about 90 pupae. They were the only lines which did not produce heterosis when they were

TABLE 30 Pupae number means of crosses between selected for pupae number, control and inbred lines at the end of the selection programme +

Matings	Means	H++	Matings	Means	H
$I_2 \times I_2$	58.40		$PSC_3 \times PSH_4$	79.63	13.11**
$I_2 \times I_3$	75.20	18.2**	$PSH_4 \times PSC_2$	72.46	
$I_3 \times I_2$	82.43				
$I_3 \times I_3$	62.76		$PSC_2 \times PLH_2$	100.86	17.06**
			$PLH_2 \times PSC_2$	91.73	
$PSC_2 \times PSC_2$	60.60				
$PSC_2 \times I_2$	86.87	23.7**	$PSH_1 \times PSH_1$	63.86	
$I_2 \times PSC_2$	79.63		$PSH_1 \times PSH_4$	74.10	13.50**
			$PSH_4 \times PSH_1$	82.03	
$PSH_4 \times PSH_4$	65.26				
$PSH_4 \times I_2$	86.90	27.32**	$PSH_4 \times PLH_2$	101.46	17.72**
$I_2 \times PSH_4$	91.40		$PLH_2 \times PSH_4$	87.10	
$PLH_2 \times PLH_2$	97.86		$PLH_1 \times PLH_1$	85.60	
$PLH_2 \times I_2$	88.53	13.76**	$PLH_1 \times PLH_2$	99.66	1.93
$I_2 \times PLH_2$	95.28		$PLH_2 \times PLH_1$	87.66	
$PSC_3 \times PSC_3$	87.06				
$PSC_3 \times PSC_2$	91.43	12.85**			
$PSC_2 \times PSC_3$	81.93				

+ Each mean has 30 observations. The population mean was 79.46 and the standard deviation was 16.17.

++ Heterosis ** $P < .01$

crossed. Maximum heterosis was observed when small population size lines were crossed to inbred lines, it was higher than the heterosis yielded by the cross of two inbred lines. The difference was significant and in the case of control lines, only chance events can be invoked for a plausible explanation. However, in small selected lines, selection may produce fixation of some genes lost in the inbred lines and then the crossbred progeny may have been benefited by that. As in the case of body length, crosses of replicates of the same treatment yielded less heterosis than crosses of replicates of different treatments.

From fig. 22, we can see that the large population size selected lines had the highest pupae number mean. The mean parent values on the straight line show very neatly the effect of N after a long period of time on the mean of a character showing inbreeding depression. All the crosses in which large population size selected lines were involved had the largest pupae number values. From this it can be argued that population size has a role in predicting selection response in small populations even although environmental factors hamper the use of current genetic models. It is clear that after 30 generations, inbreeding is playing an important part in small populations which complicates the explanation of long term selection results in characters showing inbreeding depression such as pupae number.

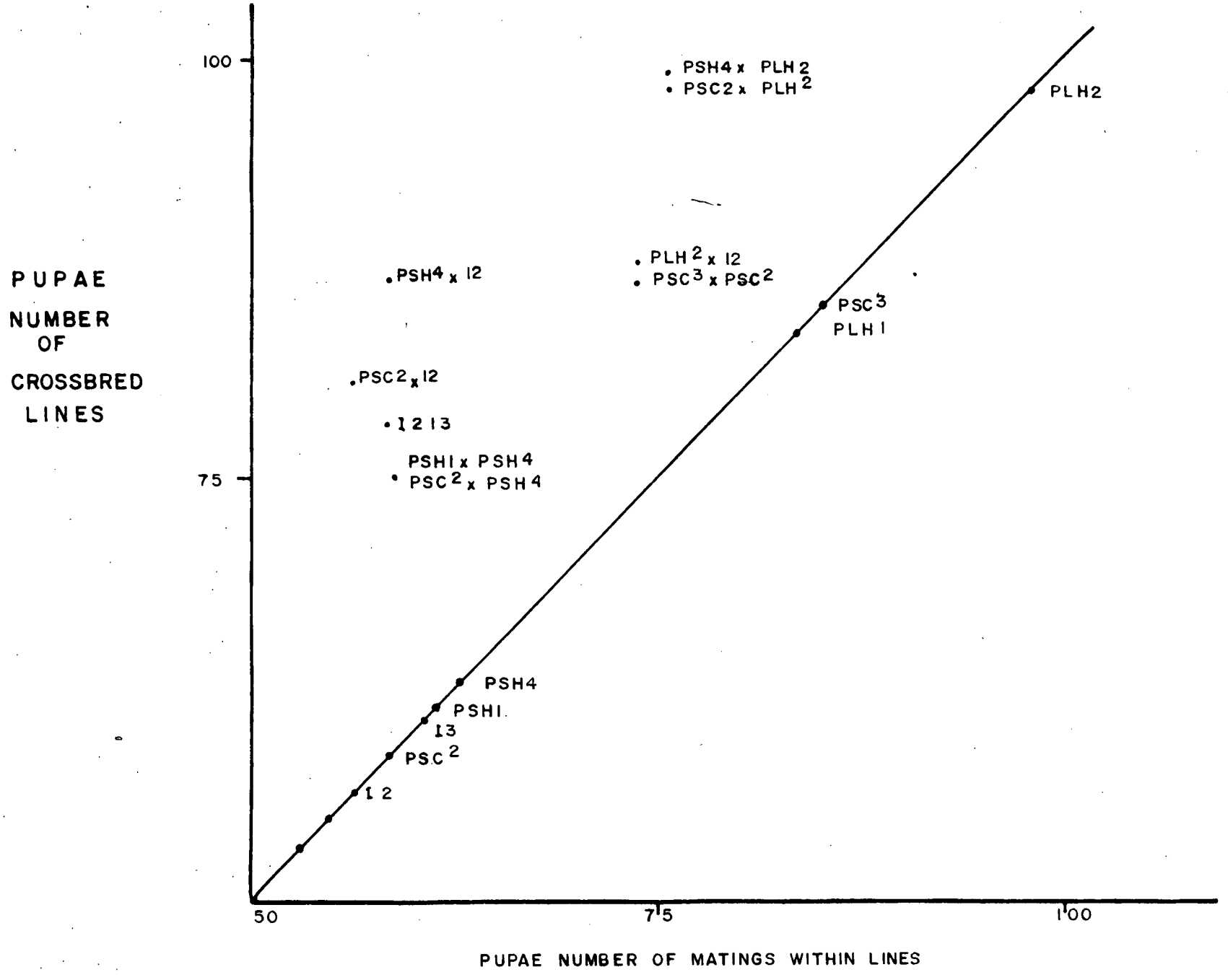


FIG. 22 HETEROSIS OF CROSSES PRESENTED IN TABLE 30.

5. Discussion

Short-term response to selection for pupae number

a) Early response to selection for pupae number was greater than expected. The lines that responded less than expectations (PLH, PLH_2, PSM_3 and PSH_3) started either with a mean close to the ceiling value (± 100 pupae) or with a high rate of response so they reached it early. Therefore they did not have any opportunity to respond further.

The high response for pupae number, something not commonly found in characters close related to reproductive fitness, may be explained by selection under sub-optimal feeding conditions followed by selection under normal conditions, it perhaps exposed to selection genetic variations not seen under normal conditions, as was indicated by Robertson F. (1960a).

b) The effect of i on short-term response to selection was not consistent. Larval competition as a result of an increase in egg production may explain that odd result.

c) Realized heritabilities increased as intensity of selection decreased. This was found as well by Frankham et al (1968) Hanrahan et al (1973) and in our results for body length. However Clayton et al (1957) reported an increase in realised heritabilities as selection intensity increased.

d) The effect of N on response to selection and realized heritability was not consistent.

e) Response to selection patterns depended on the ceiling

imposed by larval competition due to sub-optimal feeding conditions, and the initial pupae number mean or the initial rate of response. As soon as a line reached the ceiling its pattern of response became almost flat.

f) The agreement between replicates was poor. As N increased variation between replicates decreased.

g) Inbreeding perhaps did not depress the pupae number mean up to generation 10. Our inbred lines started showing inbreeding depression about generation 4 when the level of inbreeding was theoretically .60. At this point the mean pupae number had been depressed by .56p. The expected inbreeding coefficients of our small lines without selection is about .127. The selected lines would not show much greater inbreeding than this. Martin and Bell (1960) found a decline in egg production of .37 eggs and .21% in adult emergence for 1% of inbreeding. If we use these values we would expect to see a decrease in pupae number mean of about .46p (7 pupae). This is much greater than was seen in $\overline{PSC}$ lines. Our results from inbred lines agree better with the results from the $\overline{PSC}$ lines.

Long term selection response for pupae number

a) After a line reached a mean pupae production of about 100 pupae it could not go any further due to the ceiling imposed by the amount of nutrients available in our vials. Thus, what we were selecting were flies which were fast feeding larvae, that could reach their critical weight, but that had their adult body size reduced. As a consequence, their egg production was reduced. Burnet et al (1977) found that strains selected for fast feeding larvae eat faster

than unselected strains but they do not grow faster, therefore their food conversion efficiency was reduced. Thus, under a constant amount of nutrients available we cannot get more pupae by increasing the feeding speed of the larvae.

b) This brought about a negative covariance between mother and female offspring for body length and pupae number respectively, which affected both the estimations of genetic parameters and the correlated response to selection.

c) The importance of population size was shown in later generations through inbreeding. Large population size lines kept their mean pupae production about the same level imposed by the ceiling, whereas small population size lines dropped their mean pupae production by about 1.5 .

d) Heterosis was present in almost all the crosses and the only exception to this was the cross between large population size lines.

One thing of practical importance that was observed in body length by Robertson and Reeve (1955a) is that lines with more divergent means tended to produce crossbreds with an intermediate mean, whereas lines with more similar means had F.'s closely resembling the larger parent. We could not find that tendency in body length or in pupae number.

Summary

1. An experiment was carried out to study the effect of N and i on response to selection for pupae number. This was defined as the number of pupae from a 5 day egg laying period (at the peak of egg production) counted 15 days after a single pair of flies were put into a vial. The lines were initiated from the same base population as the lines selected for body length and selected over 30 generations with population sizes of 10 and 40 pairs of parents and selection intensities of 20 and 50% as well as unselected controls.
2. Early response to selection was greater than expected.
3. Realised heritabilities increased as intensity of selection decreased.
4. The effect of N on response to selection and realized heritability was not consistent.
5. The agreement between replicates was poor. As N increased variation between replicates decreased.
6. Inbreeding did not appear to depress the pupae number mean in the short-term period of this experiment.
7. After a line reached a mean pupae production of about 100 pupae it could not increase any further due to the ceiling imposed by the amount of nutrients available in our vials.
8. That ceiling created larvae competition which brought about:

a) A reduction of larvae survival

b) A reduction of adult body size

c) And as a consequence a reduction of egg production

d) Time to eclosion was prolonged.

9. It was argued that larval competition generated a negative covariance between body length of the mother and pupae number of the female offspring.

10. Inbreeding by fullsib mating reduced pupae number mean at a rate of .16 pupae per 1% of inbreeding.

11. When selected lines were crossed heterosis was shown by all the crosses with the exception of the cross between large population size lines.

VI Correlated reponse to selection

1. Introduction

Selection for a trait changes a population mean as a consequence of the change in the frequency of the genes affecting it. As genes may have pleiotropic effects or be linked to genes affecting other traits, selection for a trait may change the population mean of those other traits. This is called a correlated response to selection.

Correlated response to selection has been observed and measured in selection programmes. The theory predicting correlated responses in large populations and for a few cycles of selection (strictly one) is well known (Lerner 1950, chapter 12; Falconer 1960, chapter 19) and has been tested experimentally (Falconer 1954, Siegel 1962, Verghese et al 1968, Bell and Burris 1973 etc.) They found fair agreement with expectations or at least in the same direction. However asymmetry in correlated responses has often been found. In fact Bohren et al (1966) showed theoretically and found in their simulations in computer studies, that it must be rather surprising to find symmetry in correlated responses and that loci contributing negatively to the covariance and having frequencies other than a half seem to be the most frequent cause of this.

It is expected that in small breeding populations effective population size and intensity of selection will affect the correlated responses. Changes in gene frequencies either by drift or selection would affect the additive covariance more than the additive variance (Bohren et al 1966). Eisen et al

(1973) found, when selecting mice for postweaning gain, that the effects of population size and selection intensity on the correlated responses were in agreement with what they found in direct responses in small populations: correlated responses in the body weight traits and litter size increased as selection intensity increased and as effective population size increased. However very small population size treatments had some negative correlated responses in spite of the positive genetic correlation in the base population. They suggested it was due to inbreeding depression.

Traits closely associated with fitness have been followed in selection experiments and their correlated responses due to selection for the primary traits have been assessed. To explain the reduction in fitness of populations undergoing artificial selection for a metric character (which commonly occurs) two kind of models have been used. The metric deviation or optimum models and the homeostatic or heterotic models (Robertson 1956 and Lewontin 1964 a, b).

The former relates reproductive fitness directly to the phenotype for the metric character, irrespective of the underlying genome. Fitness declines as the square of the deviation of the metric phenotype from some fixed optimum or the population mean. In the latter model in which extreme metric deviants are less fit because they are more homozygous, fitness will decline as the square of the deviation from the mean phenotypic value of heterozygotes at which fitness will be at a maximum.

Mather and Harrison (1949) attributed the decline in

fertility of their abdominal bristle line of Drosophila melanogaster mainly to linkage. They clearly stated "Whatever drift there may be and under whatever restricted circumstances it may occur, linkage of the genes must always be causing the characters to push one another about, the trend in any one, relative to its optimum level of expression depending on the strength of the selection under which it finds itself relative to the strength of selection acting on the others". Latter and Robertson (1962) suggested linkage as the most likely explanation for the performance of two of their lines selected for abdominal bristle number. The reduction in fitness of lines selected for this trait was in accord with the theory that suggests that the decline in fitness is proportional to the square of the advance under artificial selection. However selection for body length do not appear to behave in this way. In both traits, inbreeding resulting either from drift due to small effective population size or selection itself caused a substantial decline in the mean.

Robertson and Reeve (1952) selected Drosophila flies for long and short wings. The former did not reduce either the percentage of emergence nor egg production whereas the latter did. They found three lethal factors in the second and third chromosomes of the short wing line. Furthermore they suggested the possibility that these lethal factors might be linked to alleles causing reduction in body size.

Litter size of mice selected for body weight was not affected by selection in either direction (Falconer and King 1953). They suggested as an explanation, that genetic correlation

might depend on the pleiotropic action of a few genes only, and these genes may have become fixed during the early stages of selection. Wallinga and Bakker (1978) did not find correlated response in body weight when they selected mice for litter size but generally selection for body weight has increased litter size, (Falconer 1953, Rahnefeld et al 1963, Legates 1969, Eisen et al 1973). Verghese and Nordskog (1968) evaluated correlated responses in reproductive fitness of lines selected for body and egg weight in chickens. Both the homeostatic and the optimum model would explain the decline in fitness of the selected lines. Indeed as Nicholas and Robertson (1980) pointed out there seems to be no aspect of observable response to artificial selection which would allow a distinction to be made between the two models. They commented that it permitted James (1962) to explain Lerner and Dempster's (1951) data using the optimum model.

Eisen et al (1973) attributed the fitness decline in their large effective population size lines to natural selection moving the population mean of body weight away from an optimum.

The interaction of artificial and natural selection is an interesting issue far from being fully understood in spite of the theoretical and experimental work devoted to it (see for a review Nicholas 1974 and Nicholas and Robertson 1980).

2. Methods

2.1 Estimation of correlated responses

Correlated responses were measured in generations 5, 10 and at the end of the selection programme. A sample of 30 single pair matings was set up in fresh food vials for each

body length line using randomly collected virgin flies. These were scored for pupae number. Similarly a sample of 20 males and 20 females from each pupae line was taken at random and their thorax length was measured. Care was taken that flies were picked evenly from all the vials in which the members of that line were kept. The same procedures as for the direct selected characters were used to make those measurements. Correlated responses will be presented for generations 5, 10 and at the end of the selection programme as absolute values of treatment means, and control values will also be given.

Correlated responses of lines at generation 10 will be presented as the mean value observed at that generation deviated from their own controls and divided by ten. This will be used to compare with the expected response. This was calculated using parameters in the initial base population. Correlated responses at the end of the selection programme will be presented as the mean value observed at that time, deviated from control.

2.2 Estimation of genetic parameters. Genetic correlations for all the lines were calculated at generation 5, 10 and at the end of the selection programme. Fifty pairs of flies were used for each progeny testing programme following Reeve's (1953) and Hill's (1970) methods of estimating heritabilities and genetic correlations of two characters were used. The extreme 10 pairs were selected and assortatively mated. Three males and three females offspring for each mating were measured for body length and pupae production of 6 female offspring was scored. All the technical procedures were as

explained in the Research Programme chapter.

Standardized correlated responses were estimated for each treatment by using the following formulae:

$CR'_{P.B} = CR_{P.B}/i_B\sigma_P$ and $CR'_{B.P} = CR_{B.P}/i_P\sigma_B$ where $CR'_{P.B}$ and $CR'_{B.P}$ stands for standardized correlated response for pupae number when selection is for body length and standardized correlated response for body length when selection is for pupae number respectively; $CR_{P.B}$ and $CR_{B.P}$ are the respective correlated responses, and i_B and i_P are the standardized selection differentials for body length lines and pupae number lines respectively. Standardized correlated responses were used to assess asymmetry of the correlated responses.

Realised genetic correlations were estimated using the direct and the correlated responses applying the formulae

$$\hat{r}_g = \frac{CR_{P.B}}{R_B}\,\frac{\sigma_{gB}}{\sigma_{gP}} = \frac{CR_{B.P}}{R_P}\,\frac{\sigma_{gP}}{\sigma_{gB}}$$

Heterosis in the secondary character was estimated in all the crosses carried out to estimate heterosis in the primary character.

3. Results

3.1 Short term correlated response in body lines.

Correlated responses were the means of the secondary character at generation ten deviated from the control and divided by ten. These values were taken as the average correlated response in this period. With this limitation and knowing that the sampling errors might be large, much care

has to be taken when considering these results and the conclusions drawn from them. The correlated response in pupae number in the body selected lines followed the tendency shown by the primary character. As population size and intensity of selection increased $CR_{P.B}$ increased too. (See table 31).

Table 31. Correlated responses in pupae number of body length selection lines ($CR_{P.B}$) during the 10 first generations. *(Standard errors of the line averages were calculated using between replicate variance).

Body lines	$CR_{P.B}$	Body lines	$CR_{P.B}$
BSM_1	-.031	BSH_1	-1.120
BSM_2	-.042	BSH_2	-2.220
BSM_3	.062	BSH_3	.520
BSM_4	-.413	BSH_4	-.520
$\overline{BSM}$	-.207 ± .10	$\overline{BSH}$	-.830 ± .57
$\widehat{BSM}$	-1.782	$\widehat{BSH}$	-3.096
BLM_1	.421	BLH_1	-1.360
BLM_2	-.989	BLH_2	.900
$\overline{BLM}$	-.243 ± .71	$\overline{BLH}$	-.320 ± 1.14
$\widehat{BLM}$	-1.848	$\widehat{BLH}$	-3.252

$\widehat{\ }$ stands for expected correlated response, calculated using parameters from the base population at generation O.

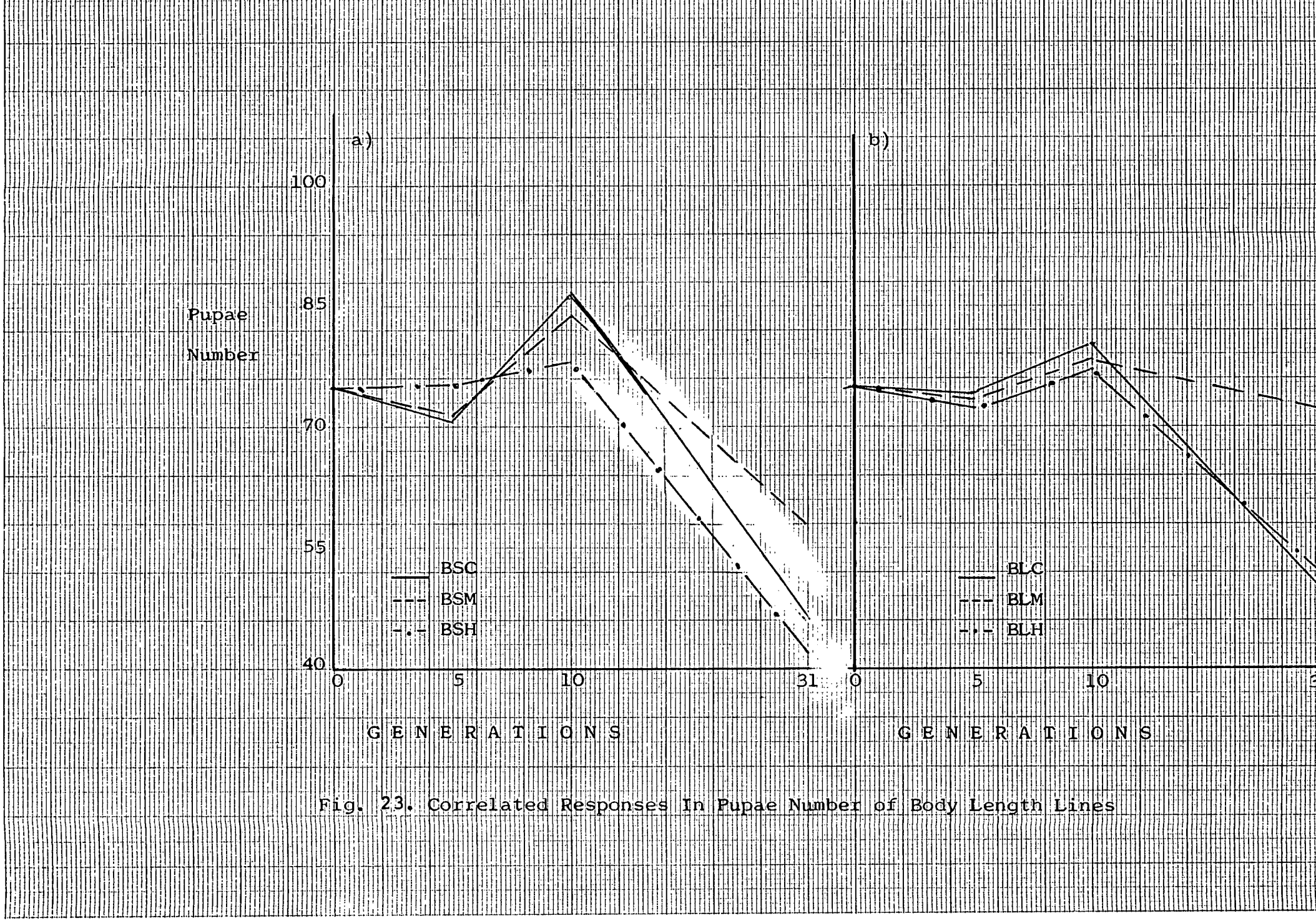


Fig. 23. Correlated Responses In Pupae Number of Body Length Lines

Poor agreement was found between replicates. Observed correlated responses were smaller than expectations. Correlated responses of high selection intensity lines were more different from predictions than those of intermediate intensity of selection lines. This pattern was observed in the direct response as well.

From Fig. 23 a, b it can be seen that pupae number in body lines had its mean increased. The increase in selected lines, however, was less than in controls.

3.2 Short term correlated response in pupae lines

Correlated responses for body length in pupae lines were in the opposite direction to predictions, but not far from zero, (see table 32).

Table 32. Correlated responses for body length of pupae number lines ($CR_{B.P}$) during the 10 first generations.

Pupae lines	$CR_{B.P}$	Pupae lines	$CR_{B.P}$
PSM_1	.065	PSH_1	.108
PSM_2	.141	PSH_2	.392
PSM_3	.134	PSH_3	-.015
PSM_4	.055	PSH_4	-.048
$\overline{PSM}$	.088 ± .02	$\overline{PSH}$	.088 ± .10
$\widehat{PSM}$	-.187	$\widehat{PSH}$	-.328
PLM_1	-.045	PLH_1	.020
PLM_2	-.165	PLH_2	.062
$\overline{PLM}$	-.100 ± .06	$\overline{PLH}$	.041 ± .02
$\widehat{PLM}$	-.191	$\widehat{PLH}$	-.339

$\widehat{\ }$ stands for expected correlated response calculated using parameters from the base population at generation O. Standard errors of the correlated response average was calculated using the between replicate variance.

The effect of N and i on correlated response was not consistent. This was observed in the primary character as well. However there was a tendency for the correlated response to increase as the direct response increased.

Fig. 24 shows us that body length of pupae lines increased in this short-term period.

If we look at Fig. 24a and 24b we can see a tendency of changing body length correlated to a change in pupae number. This was shown by the controls and the selected lines. Therefore it is likely that environmental correlations are involved in the correlated responses to selection.

3.3 Standardized correlated responses

Standardized correlated responses are presented in Table 33. Comparing values of body and pupae lines within the same treatment it is clear that there is a tendency to asymmetry in the correlated responses.

Table 33. Standardized correlated responses ($CR'_{P.B.}$, $CR'_{B.P}$) of body and pupae lines during the first 10 generations.

Body lines	$CR'_{P.B.}$	Pupae lines	$CR'_{B.P}$
$\overline{BSM}$	-.027	$\overline{PSM}$	.081
$\overline{BSH}$	-.149	$\overline{PSH}$	.041
$\overline{BLM}$	-.015	$\overline{PLM}$	-.114
$\overline{BLH}$	-.011	$\overline{PLH}$	.131

3.4 Realised genetic correlations ($\hat{r}_g$). Table 34 presents $\hat{r}_g$ values for body and pupae lines. The realized genetic correlations calculated from the correlated response in pupae number

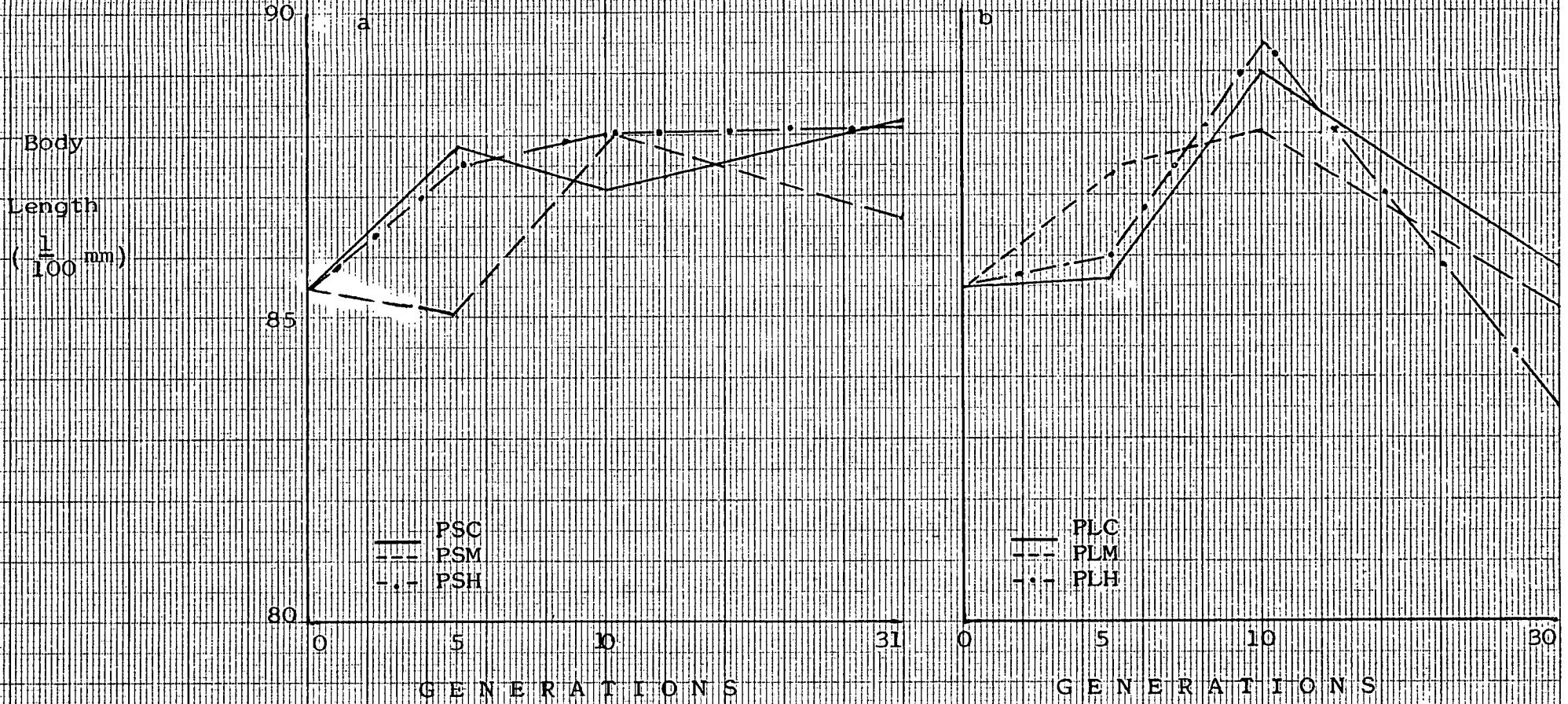


Fig. 24 Correlated Responses in Body Length of Pupae Number Lines.

Table 34 Realized genetic correlations ($\hat{r}_g$) in body and pupae lines during the first 10 generations.

Body lines	$\hat{r}_g$	Pupae lines	$\hat{r}_g$
BSM_1	-.015 ± .05	PSM_1	.492 ± .06
BSM_2	-.017 ± .05	PSM_2	.290 ± .06
BSM_3	.045 ± .05	PSM_3	4.214 ± .06
BSM_4	-.263 ± .05	PSM_4	.095 ± .06
$\overline{BSM}$	-.112 ± .03	$\overline{PSM}$	.347 ± .68
BSH_1	-.335 ± .03	PSH_1	.324 ± .03
BSH_2	-.940 ± .03	PSH_2	1.071 ± .03
BSH_3	.320 ± .03	PSH_3	-.113 ± .03
BSH_4	-.226 ± .03	PSH_4	-.159 ± .03
$\overline{BSH}$	-.374 ± .25	$\overline{PSH}$	.305 ± .640
BLM_1	.166 ± .02	PLM_1	-.089 ± .03
BLM_2	-.899 ± .02	PLM_2	-.429 ± .03
$\overline{BLM}$	-.127 ± .53	$\overline{PLM}$	-.227 ± .17
BLH_1	-.695 ± .01	PLH_1	.479 ± .02
BLH_2	.238 ± .01	PLH_2	-15.98 ± .02
$\overline{BLH}$	-.127 ± .47	$\overline{PLH}$	2.03 ± ?

Standard errors were calculated using Hill's (1971) formulae for the replicate lines and the between replicate variance for the lines average.

of body lines are in general negative but much closer to zero than the genetic correlation estimated in the base population (-.954). Three of the average values are rather similar. However there is poor agreement between replicates. The realized genetic correlations calculated from the correlated response in body length of pupae lines are in general positive. Only PLM lines had consistent negative realized genetic correlations. There is, very poor agreement between replicates.

3.5 Genetic correlation estimates.

At generation 5, 10 and at "the limit" genetic correlations between body length and pupae number were estimated in body length and pupae number selection lines. They are presented in the appendix. They have large sampling errors. They suggest that the genetic correlation estimated in the base population was overestimated. However, we feel that the genetic correlation is negative and much closer to zero than that estimated; and that the genetic correlation did not change a lot during the selection programme.

3.6 Correlated response when selection was stopped.

In Table 35 correlated and direct responses at the end of the selection experiments are presented. It is interesting to see that lines which responded more to direct selection had more negative correlated responses.

Table 35. Direct and correlated responses at the end of the selection experiments.

Body selected lines			Pupae selected lines		
	+Body length	+Pupae number		Body length	Pupae number
$\overline{BSM}$	11.0	11.9	$\overline{PSM}$	-2.4	26.10
$\overline{BSH}$	14.8	-5.2	$\overline{PSH}$	-.04	23.36
$\overline{BLM}$	8.8	22.5	$\overline{PLM}$	-1.18	23.66
$\overline{BLH}$	13.8	2.0	$\overline{PLH}$	-4.50	27.14

+ Average of mean deviate from controls.

Although only one average ($\overline{BSH}$) showed a negative correlated value, treatments with more response for body length had lower pupae number means. This can be seen as well in the pupae lines in which treatments that yielded larger pupae number values had lower body length means.

Correlated responses followed the pattern of direct selection responses in the opposite direction and this reinforces the point that selection at the limit is affected by population size not only because of inbreeding but because of chance fixation of undesirable genes. Otherwise we could not explain why $\overline{BLH}$ had a lower pupae number than $\overline{BSM}$ which has a higher level of inbreeding not only because of its smaller population size but because its experimental life was longer.

3.7 Heterosis in the secondary character in crosses at the end of the selection programme.

Heterosis in pupae number in crosses between body lines and inbred lines is shown in table 36. Some environmental factor had depressed pupae production mean. This becomes clear if we compare

Table 36 - Pupae number means of crosses between selected body length, control and inbred lines at the end of the selection programme.

Matings	$\bar{X}$	H^+	Matings	$\bar{X}$	H
$I_2 \times I_2$	45.60		$BSM_4 \times BSH_2$	46.13	5.03*
$I_2 \times I_3$	41.00	-5.80*	$BSH_2 \times BSM_4$	42.70	
$I_3 \times I_2$	52.22				
$I_3 \times I_3$	59.22		$BSH_2 \times BSH_1$	46.73	
			$BSH_1 \times BSH_2$	45.66	1.84
$I_2 \times BSC_2$	44.60		$BSH_1 \times BSH_1$	50.60	
$BSC_2 \times I_2$	65.06	7.31**			
$BSC_2 \times BSC_2$	49.43		$BSH_3 \times BSH_3$	45.10	
			$BSH_3 \times BSH_4$	37.53	-4.68*
$I_2 \times BSM_4$	50.86		$BSH_4 \times BSH_3$	49.16	
$BSM_4 \times I_2$	71.20	17.90**	$BSH_4 \times BSH_4$	50.96	
$BSM_4 \times BSM_4$	40.66				
			$BSH_2 \times BLM_2$	63.50	
$I_2 \times BSH_2$	58.16		$BLM_2 \times BSH_2$	44.60	6.62**
$BSH_2 \times I_2$	55.93	15.19**	$BLM_2 \times BLM_2$	56.76	
$BSH_2 \times BSH_2$	38.10				
$BSC_2 \times BSM_4$	58.66	13.61**			
$BSC_2 \times BSH_2$	40.10	-3.66			

^+H Heterosis in the crosses

* and ** statistical significance at .05 and .01 respectively

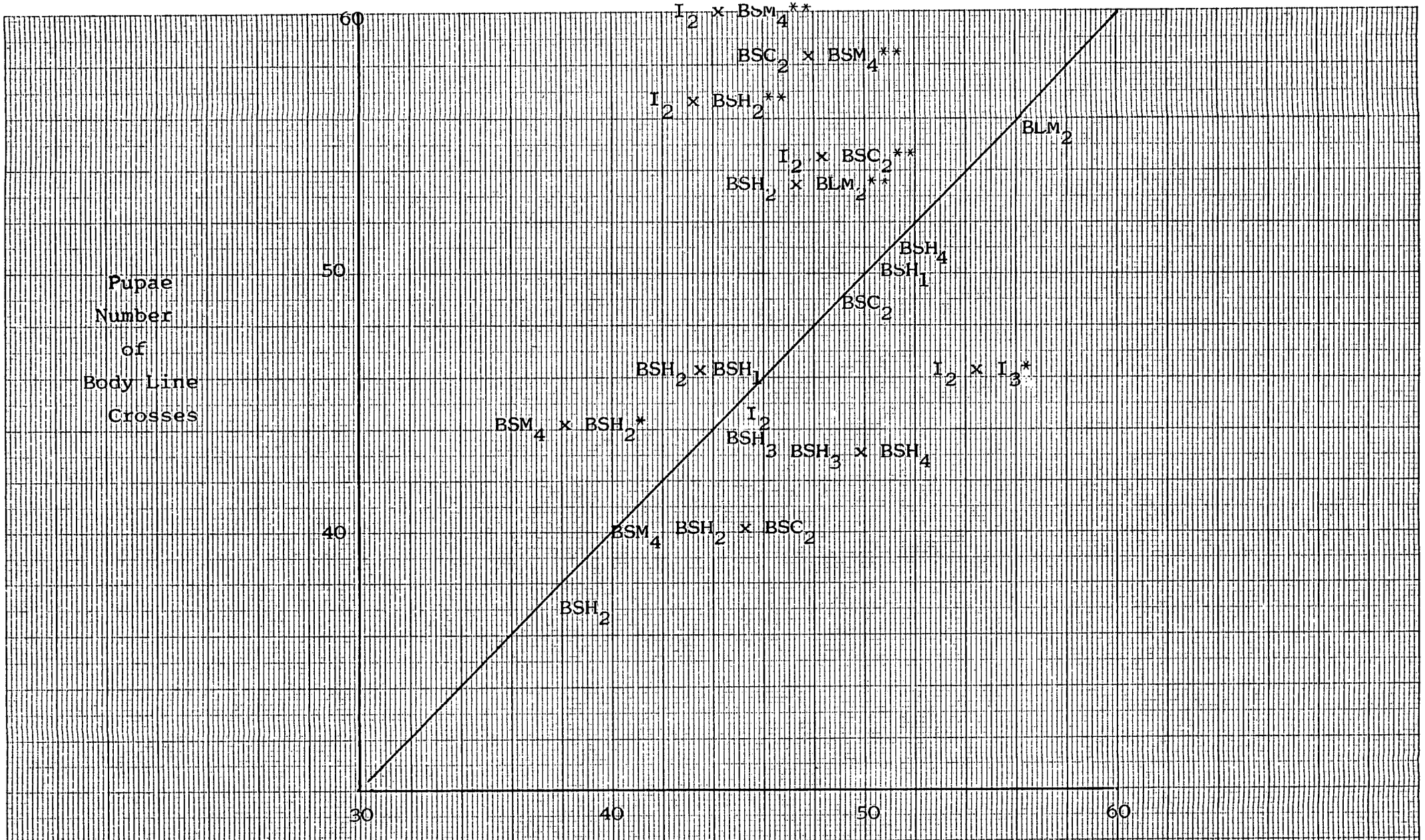


Fig.25 Pupae Number Heterosis In Body Line Crosses.

the mean parental value of inbred lines of this table with that in table 30 when the same lines were crossed together with pupae lines. Heterosis was present in most of the crosses, being higher for those in which inbred lines and small population size lines were involved. Crosses between inbred lines gave negative heterosis and that is hard to explain as the same cross a week afterwards yielded positive heterosis. The pupae number means of crosses between replicate lines are smaller than those between inbred lines. They gave the highest heterosis when crossed with inbred or small population size control lines. Crosses between replicates of body lines within treatments yielded no or negative heterosis for pupae number though they had yielded a fair amount of heterosis for body length.

In general crosses that gave high heterosis for body length showed high heterosis for pupae number too. This can be seen clearly from figs. 14 and 25. Heterosis for body length in crosses between pupae lines and inbred lines are shown in table 37 and Fig. 26. Crosses of inbred lines with small population pupae lines yielded heterosis in body length. However when an inbred line was crossed with a large population size line no heterosis was found. Matings between replicates of the same treatment showed no heterosis. All body length means of within line matings of pupae lines were below the mean value of inbred lines except PLH_1 (See Fig. 26).

PLH_1 line had a low mean body length during selection but after one generation of random mating its mean pupae number

Table 37. Body length means of crosses between selected pupae number, control and inbred lines at the end of the selection programme.

Matings	$\bar{X}$	H^+	Matings	$\bar{X}$	H
$I_2 \times I_2$	91.07		$PSC_2 \times PSC_3$	90.52	
$I_2 \times I_3$	92.21	1.19**	$PSC_3 \times PSC_2$	90.74	.61
$I_3 \times I_2$	93.35		$PSC_3 \times PSC_3$	90.52	
$I_3 \times I_3$	92.10				
			$PSH_4 \times PSH_1$	90.92	
$I_2 \times PSC_2$	91.60		$PSH_1 \times PSH_4$	90.67	.11
$PSC_2 \times I_2$	95.10	3.05**	$PSH_1 \times PSH_1$	90.42	
$PSC_2 \times PSC_2$	89.52				
			$PLH_2 \times PLH_1$	91.35	
$I_2 \times PSH_4$	93.21		$PLH_1 \times PLH_2$	91.07	.62
$PSH_4 \times I_2$	92.60	1.89**	$PLH_1 \times PLH_1$	91.52	
$PSH_4 \times PSH_4$	90.95				
$I_2 \times PLH_2$	90.35				
$PLH_2 \times I_2$	90.71	.14			
$PLH_2 \times PLH_2$	89.67				

^+H heterosis in the crosses

** statistical significance at .01

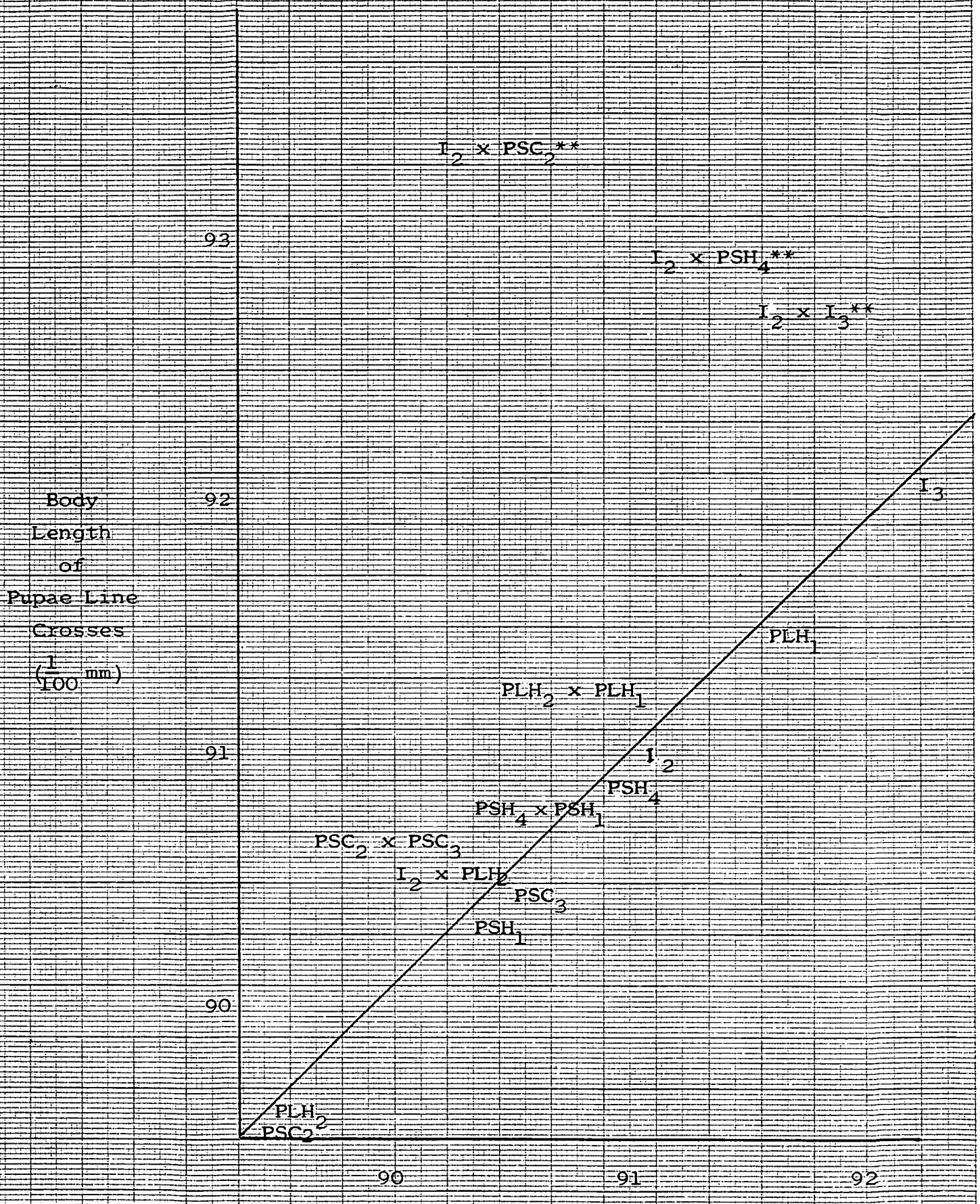


Fig. 26. Body Length Heterosis in Pupae Line Crosses

declined and the body length mean increased. It can be argued that natural selection for heterozygous loci in which alleles that increase body length decrease pupae number mean, was interacting with our selection programme.

4. Discussion

4.1 Short-term correlated responses

Correlated responses for pupae number in body length selection lines.

a) Correlated responses had a tendency to be more negative as N and i increased. This was found also in the primary character and it agrees with Eisen et al (1973) results.

b) Observed correlated responses were of the same sign as but smaller than expectations. Robertson and Reeve (1952) we have seen, found no reduction in egg production nor in percentage of emergence in their long wing line.

There were several observations that suggest r_g calculated in the base population was overestimated. Furthermore, an improvement of the environment, causing increase of absolute value of pupae number means, may create a positive correlation between body length and egg production, as was found by Robertson F. (1966) and this might have obscured the underlying negative genetic correlation.

Correlated response for body length in pupae number selection lines

a) Pupae number lines had a positive correlated response for body length. It can be argued that the phenotypic correlation

generated by suboptimal feeding conditions would have been magnified by stronger larval competition as a result of an increase in egg production. The only line average which showed a negative correlated response ($\overline{PLM}$) was the one that did not have its mean pupae number around the ceiling imposed by larval competition.

b) The effect of N and i were not consistent. It seems that other forces were affecting body length in pupae selection lines which obscured the effect of selection and population size.

Standardized correlated responses.

Standardized correlated responses showed a great asymmetry for these characters. We have mentioned the abundance of reports of this phenomenon in the literature. The occurrence of asymmetry has been shown to be highly probable (Bohren et al 1966). The asymmetry we found would suggest that the relationship between body length and pupae number was altered by the environment the flies lived in, but mainly during their larval period as was indicated by Robertson F. (1963).

4.2 Long term correlated responses

Correlated responses for pupae number in body length selection lines.

a) Pupae number means of body selection lines decreased at the end of the selection programme. Inbreeding played an important role in this result, but nevertheless some environmental factor might have depressed pupae production. The decrease of pupae production in $\overline{BLC}$ can not be explained by inbreeding depression only. The presence of lethals as argued by Reeve and Robertson (1953) can account in part for this result as well.

This explains why only one average line ($\overline{BSH}$) yielded a negative correlated response.

b) Nevertheless, the effect of N and i agrees with early results. Eisen (1975) found the effect of N and i in long-term correlated response to selection consistent and in agreement with direct response.

Correlated responses for body length in pupae number selected lines.

a) The effect of i on correlated response for body length was not consistent. An increase in response of pupae number should have produced a reduction of body length. This is why the effect of i on correlated response agrees with the direct response.

b) The effect of N on correlated response was consistent. As N increased the correlated response increased at both levels of i.

These results warn us of the need for a better understanding of environmental effects such as larval competition, as they can lead us to draw very wrong conclusions about theoretical models proposed to explain real life results. This has also been the case with maternal effects in mice.

Crossing of selected lines.

a) Small population size body length selection lines yielded higher heterosis for pupae number when crossed with small control lines or inbred lines than crosses between small control and inbred lines. This suggests that genes with negative pleiotropic effect on body length and pupae number were fixed in

the small population body length selection lines. Robertson and Reeve (1952) argued the presence of genes reducing fertility and viability linked with the genes favouring body length.

b) The behaviour of correlated characters when selected lines are crossed has practical consequences. In our lines selected for body length pupae production was decreased. However, when they were crossed pupae production recovered. Heterosis for body length and pupae number was shown by those crosses. Their pupae production was higher than the best parent.

VII General Discussion

Short-term response to selection reflected the genetic parameters estimated in the base population. However it appeared that the estimated negative genetic correlation was an overestimate of the real one. Pupae number selection lines were very much affected by environmental factors and to explain the results we have to invoke that the genetic properties of these lines, as genetic variation, genetic correlation between body size and fertility were all altered as a consequence of environmental changes. Robertson (1960a), Sang (1962) and our secondary experiments also showed this phenomenon.

The effect of N on absolute responses and the realised heritabilities proved to be an important factor to consider in short-term selection. Even a character such as body length of Drosophila, very little affected by inbreeding depression, showed less response when selection was carried out in a smaller population. This effect has been observed in several selection experiments with different species of animals as mentioned earlier. The correlated responses were in agreement with direct responses, as was found by Eisen *et al* (1973).

Realised heritabilities in both body length selection lines and pupae number selection lines decreased as i increased. This has been reported by Frankham *et al* (1968) and Hanrahan *et al* (1973), however Clayton *et al* (1957) found an increase in realised heritability as i increased.

Variation in response to selection between replicates has

been a common observation in replicated selection experiments. We would suggest that the initial sampling plays an important role in this result, as our data indicated.

Long-term selection response showed the importance of N in influencing the response to selection given the genetic variance present in the base population. Body length selection lines with small Ni yielded results which agreed well with Robertson's (1960) theory. This was found by Jones et al (1968) and Eisen (1975) as well. Larger values of Ni gave response to selection far from 2N times the response in the first generation. We should point out as Jones et al (1968) and Eisen (1975) did the possibility of further improvement in those lines. Linkage of genes at low frequency would affect more response to selection of treatments with larger Ni as was indicated by Hill and Robertson (1966). James (1962 and 1965) pointed out that other forces such as natural selection might affect long-term responses more for larger values of N. The reduction of response due to natural selection might have been of different nature. $\overline{BLH}$ lines had their pupae number reduced as a correlated response. It is suggested as a result an increasing of gene frequency/ of alleles with negative pleiotropic effect on body length and pupae number. When selection was relaxed they lost almost half of their gain in body length and pupae production was recovered. In these lines genetic variation in body length was still present at the end of the selection programme. They yielded very low heterosis for body length and pupae number when they were crossed. In $\overline{BLM}$ lines selection for lethals in the heterozygote state could account for the early reduction in rate of response. This was argued by Robertson and Reeve (1952)

as well, when selecting for body size in Drosophila. In these lines pupae number did not reduce during the selection programme. Genetic variation still was present at the end of the selection programme and they showed low heterosis for body length and pupae number when they were crossed. On the other hand small population size lines $\overline{BSH}$ and $\overline{BSM}$ suggested that they had their genetic variation exhausted. They yielded a high amount of heterosis for both characters when they were crossed.

Long-term response to selection in pupae lines was almost nil due to the ceiling imposed by larval competition. Under these circumstances the effect of N and i are only a reflection of events happened before that ceiling was reached. However the effect of N was observed through inbreeding depression.

References

Adolph, E.F. (1920). Egg-laying reactions in the Pomace fly, Drosophila. Jour. Exp. Zool. 31, 327-341.

Bakker, H., Wallinga, J.H. and Politick, R.D. (1978). Reproduction and Body Weight of mice after long-term selection for large litter size. J. of Anim. Sci. 46, 1572-1582.

Bakker, K. (1961). An analysis of factors which determine success in competition for food among larvae of Drosophila melanogaster. Archives Neerlandaises de Zoologie. 14, 200-281.

Bell, A.E. and Burris, M.J. (1973). Simultaneous selection for two correlated traits in tribolium. Genet. Res. 21, 29-46.

Bohren, B.B., Hill, W.G. and Robertson A. (1966). Some observations on asymmetrical correlated response to selection. Genet. Res. 7, 44-57.

Burnett, B., Sewell, D. and Bos, M. (1977). Genetic analysis of larval feeding behaviour in Drosophila melanogaster. II Growth relations and competition between selected lines. Genet. Res. 30, 149-161.

Clayton, G.A., Morris, J.A. and Robertson A. (1957). An experimental check on quantitative genetical theory. I. Short-term responses to selection. J. Genetics 55, 131-151.

Clayton, G.A. and Robertson A. (1957). An experimental check on quantitative genetical theory. II The long-term effects of selection. J. Genetics 55, 152-170.

Clayton, G.A., Night, B.R.K., Morris, J.A. and Robertson, A. (1957). An experimental check on quantitative genetical theory. III Correlated responses. J. Genetics 55, 171-180.

Crow, J.F. (1954). Breeding structure of populations II. Effective population number. In Statistics and Mathematics in Biology, Kempthorne et al. Ed., Iowa State College Press, Ames, Iowa. Pp. 543-556.

Crow, J.F. and Kimura, M. (1971). The effective number of a population with overlapping generations: A correction and further discussion. Amer. J. Human Genetics.

Crow, J.F. and Morton, N.E. (1955). Measurement of gene frequency drift in small populations. Evolution 9, 202-219.

Curnow, R.N. and Baker, L.H. (1968). The effect of repeated cycles of selection and regeneration in populations of finite size. Genet. Res. 11, 105-112.

Church, R.B. and Robertson F.W. (1966). Biochemical analysis of genetic differences in the growth of Drosophila. Genet. Res. 383-407.

Dalton, D.C. and Bywater, T.L. (1963). The effect of selection for litter size and litter weight at weaning in mice maintained on two diets. Anim. Prod. 5, 317-326.

Dempster, E.R. (1955). Genetic models in relation to animal breeding problems. Biometrics 11, 535-536.

Dempster, E.R. (1958). The fate of mutations in closed populations: in Proc. 11th Pacific Poultry Breeders Roundtable, University of California, California: Davis.

Dempster, E.R., Lerner, L.M. and Lowry, D.C. (1952). Continuous selection for egg production in poultry. Genetics 37, 693-708.

Dickerson, G.E. (1955). Genetic slippage in response to selection for multiple objections. Cold Spring Harb. Symp. Quant. Biol. 20, 166-177.

Eisen, E.J. (1972). Long-term selection response for 12-day litter weight in mice. Genetics 72, 129-142.

Eisen, E.J. (1975). Population size and selection intensity effects on long-term selection response in mice. Genetics 79, 305-323.

Eisen, E.J., Hanrahan, J.P. and Legates, J.E. (1973). Effects of population size and selection intensity on correlated response to selection for postweaning gain in mice. Genetics 74, 157-170.

Falconer, D.S. (1953). Selection for large and small size in mice. J. Genetics 51, 470-501.

Falconer, D.S. (1954). Validity of the theory of genetic correlation. J. Heredity 45, 42-44.

Falconer, D.S. (1955). Patterns of response in selection experiments with mice. Cold Spring Harb. Symp. Quant. Biol. 20, 178-196.

Falconer, D.S. (1960). Introduction to quantitative genetics. Edinburgh. Oliver and Boyd. Pp 365.

Falconer, D.S. and King, J.W.O. (1953). A study of selection limits in the mouse. J. Genetics 51, 561-580.

Felsenstein, J. (1965). The effect of linkage on directional selection. Genetics 52, 349-363.

Felsenstein, J. (1969). The effective size of a population with overlapping generations. Genetics 61, 18.

Frahm, R.R. (1965). Comparison of response to selection for body weight under divergent larval density conditions in Drosophila pseudoobscura. Ph.D. Thesis. North Carolina State University.

Frankham, R. (1980a). Origin of genetic variation in selection lines. Proc. of a Symposium on selection experiments in laboratory and domestic animals. Harrogate; Commonwealth Agricultural Bureaux pp. 56-68.

Frankham, R. (1980b). The founder effect and response to artificial selection in Drosophila. Proc. of a Symposium on selection experiments in laboratory and domestic animals. Harrogate; Commonwealth Agricultural Bureaux pp. 87-90.

Frankham, R., Jones, L.P. and Barker, J.S.F. (1968). The effects of population size and selection intensity in selection for a quantitative character in Drosophila. I. Short-term response to selection. Genet. Res. 12, 237-248.

Frankham, R., Jones, L.P. and Barker, J.S.F. (1968b). The effects of population size and selection intensity in selection for a quantitative character in Drosophila. III Analysis of the lines. Genet. Res. 12, 267-283.

Gill, J.L. (1965a). A Monte Carlo evaluation of predicted selection response. Aust. J. Biol. Sci. 18, 999-1007.

Gill, J.L. (1965b). Selection and linkage in simulated genetic populations. Aust. J. Biol. Sci. 18, 1171-1187.

Gowen, J.W. and Johnson, L.E. (1946). The mechanism of heterosis I. Metabolic capacity of different races of Drosophila melanogaster for egg production. The American Naturalist 80, 149-179.

Griffing, B. (1960). Theoretical consequence of truncation selection based on the individual phenotype. Aust. J. Biol. Sci. 13, 307-343.

Haldane, J.B.S. (1931). A mathematical theory of Natural and artificial selection. VII. Selection intensity as a function of mortality rate. Proc. Camb. Phil. Sci. 27, 131-136.

Hanrahan, J.P., Eisen, E.J. and Legates, J.E. (1973). Effects of population size and selection intensity on short-term response to selection for postweaning gain in mice. Genetics, 73, 513-530.

Hill, W.G. (1969b). The rate of selection advance for non-additive loci. Genet. Res. 13, 165-173.

Hill, W.G. (1969a). On the theory of artificial selection in finite populations. Genet. Res. 13, 143-163.

Hill, W.G. (1970). Design of experiments to estimate heritability by regression of offspring on selected parents. Biometrics 26, 566-571.

Hill, W.G. (1971). Design and efficiency of selection experiments for estimating genetic parameters. Biometrics 27, 293-311.

Hill, W.G. (1972a). Estimation of realised heritabilities from selection experiments. II Selection in one direction. Biometrics 28, 767-780.

Hill, W.G. (1972b). Probability of fixation of genes in populations of variable size. Theor. Popul. Biol. 3, 27-40.

Hill, W.G. (1974). Variability of response to selection in genetic experiments. Biometrics 30, 363-366.

Hill, W.G. (1977). Variation in response to selection. In. Proc. Intern. Conference on Quantitative Genetics. The Iowa State University Press, 343-365. Edit. Pollack E., Kempthorne, O. and Bailey, T.B.

Hill, W.G. and Robertson, A. (1966). The effect of linkage on limits to artificial selection. Genet. Res. 8, 269-294.

Hill, W.G. and Robertson, A. (1968). The effect of inbreeding at loci with heterozygote advantage. Genetics 60, 615-628.

Jackson, N. and Turner, H.N. (1972). Optimal structure for a cooperative nucleus breeding system. Proc. Aust. Soc. Anim. Prod. 9, 55-67.

James, J.W. (1962). Conflict between directional and centripetal selection. Heredity 17, 487-499.

James, J.W. (1965). Response curves in selection experiments. Heredity 20, 57-63.

James, J.W. (1971). The founder effect and responses to artificial selection. Genet. Res. 16, 241-250.

Jones, L.P., Frankham, R. and Barker, J.S.F. (1968). The effects of population size and selection intensity in selection for a quantitative character in Drosophila. II Long-term response to selection. Genetics 12, 249-266.

Kearsey, M.J. and Kojima, K. (1967). The genetic architecture of body weight and egg hatchability in Drosophila melanogaster. Genetics 56, 23-37.

Kidwell, J.F. and Kidwell, M.H. (1966). The effects of inbreeding on body weight and abdominal chaeta number in Drosophila melanogaster. Can. J. Genet. Cytol. 8, 207-215.

Kimura, M. (1957). Some problems of stochastic process in genetics. Anim. Math. Stat. 28, 882-901.

Kimura, M. (1962). On the probability of fixation of mutant genes in a population. Genetics 47, 713-719.

Kimura, M. and Crow, J.F. (1963). The measurement of effective population number. Evolution 17, 279-288.

Knight, D.R. and Robertson, A. (1957). Fitness as a measurable character in Drosophila. Genetics 42, 524-530.

Kojima, K. (1961). The effect of dominance and size of population on response to mass selection. Genet. Res. 2, 177-188.

Kress, D.D., Enfield, F.D. and Braskerud, O. (1971). Correlated response in male and female sterility to selection for pupa weight in Tribolium castaneum. Theoretical and Applied Genetics 41, 197-202.

Latter, B.D.H. (1965a). The response to artificial selection due to autosomal genes of large effect. I Changes in gene frequency at an additive locus. Aust. J. Biol. Sci. 18, 585-598.

Latter, B.D.H. (1965b). The response to artificial selection due to autosomal genes of large effect. II The effects of linkage on limits to selection in finite populations. Aust. J. Biol. Sci. 18, 1009-1025.

Latter, B.D.H. (1966). The response to artificial selection due to autosomal genes of large effect III The effect of linkage on the rate of advance and approach to fixation in finite populations. Aust. J. Biol. Sci. 19, 131-146.

Latter, B.D.H. (1969). Models of quantitative genetics variation and computer simulation of selection response, Proc. Intern. Conf. Computer Application Genetics University of Hawaii Press, 49-60 pp.

Latter, B.D.H. and Robertson, A. (1962). The effects of inbreeding and artificial selection on reproductive fitness. Genet. Res. 3, 110-138.

Latter, B.D.H. and Novitski, C.E. (1969). Selection in a finite population with multiple alleles. Limits to directional selection. Genetics 62, 859-876.

Legates, J.E. (1969). Direct and correlated responses to selection in mice pp 149-165. Genetics lectures. Volume 1. Edited by R. Bogart.

Lerner, I.M. (1950). Population Genetics and Animal Improvement. Cambridge University Press. pp.342.

Lewis, W.L. and Warwick, E.J. (1953). Effectiveness of selection for body weight in mice from inbred and outbred populations derived from common parent stocks. J. Hered. 44, 233-238.

Lewontin, R.C. (1964a). The interaction of selection and linkage I. General considerations, heterotic models. Genetics (49), 49-67.

Lewontin, R.C. (1964b). The interaction of selection and linkage II. Optimum models. Genetics 50, 757-782.

Linney, R., Barnes, B.W. and Kearsey, M.J. (1971). Variation for metrical characters in Drosophila populations. III The nature of selection. Heredity 27, 163-174.

Madalena, F.E. (1970). Studies on the limits to artificial selection. Ph.D. Thesis. University of Edinburgh.

Madalena, F.E. and Robertson, A. (1975). Population structure in artificial selection studies with Drosophila melanogaster. Genet. Res. 24, 113-126.

Martin, G.A. and Bell, A.E. (1960). An experimental check on the accuracy of prediction of response during selection. Biometrical Genetics. Ed. Kempthorne, O. 178-187. Pergamon Press, London.

Mather, K. and Harrison, B.J. (1949). The manifold effect of selection. Heredity 3, 1-52.

Nei, M. and Murata, M. (1966). Effective population size when fertility is inherited. Genet. Res. 8, 257-260.

Nicholas, F.W. (1974). Studies on artificial and natural selection. Ph.D. Thesis. University of Edinburgh.

Nicholas, F.W. and Robertson, A. (1980). The conflict between natural and artificial selection in finite populations. Theor. Appl. Genet. 56, 57-64.

Nordskog, A.W. Festing, M. and Verghese, M.W. (1967). Selection for egg production and correlated responses in the fowl. Genetics 55, 179-191.

O'Donald, P. (1970). Change of fitness by selection for a quantitative character. Theor. Appl. Biol. 1, 219-232.

Ohta, T. (1968). Effect of initial linkage disequilibrium and epistasis on fixation probability in a small population with two segregating loci. Theor. Appl. Genet. 38, 243-248.

Qureshi, A.W. (1968). The role of finite populations size and linkage in response to continued truncation selection. II Dominance and overdominance. Theor. Appl. Genet. 38, 267-270.

Qureshi, A.W. and Kempthorne, O. (1968). On the fixation of genes of large effects due to continued truncation selection in small populations on polygenic systems with linkage. Theor. Appl. Genet. 38, 249-255.

Qureshi, A.W., Kempthorne, O. and Hazel, L.N. (1968). The role of finite populations size and linkage in response to continued truncation selection I. Additive gene action. Theor. Appl. Genet. 38, 256-263.

Rae, A.J. (1974). The development of group breeding schemes. Some theoretical aspects. Sheep Farming Annual 121-127.

Rahnefeld, G.W., Boylan, W.J., Comstock, R.E. and Singh, M. (1963). Mass selection for postweaning growth in mice. Genetics 48, 1567-1583.

Reeve, E.C.R. (1953). Studies in quantitative inheritance III. Heritability and genetic correlation in progeny test using different mating systems. J. Genet. 51, 520-542.

Reeve, E.C.R. and Robertson, F.W. (1953). Studies in quantitative inheritance II. Analysis of a strain of Drosophila melanogaster selected for long wings. J. Genet. 51, 276-316.

Richardson, R.H. and Kojima, K. (1965). The kinds of Genetic viability in relation to selection response in Drosophila fecundity. Genetics 52, 583-598.

(Roberts: see after Robertson, F.W.)

Robertson, A. (1952). The effect of inbreeding on the variation due to recessive genes. Genetics 37, 189-207.

Robertson, A. (1955). Selection in Animals: Synthesis. Cold Spring Harb. Symp. Quant. Biol. 20, 225-229.

Robertson, A. (1956). The effect of selection against extreme deviants based on deviation or on homozygosis. J. Genet. 54, 236-248.

Robertson, A. (1960). A theory of limits in artificial selection. Proc. Roy. Soc. B. 153, 234-249.

Robertson, A. (1961). Inbreeding in artificial selection programmes. Genet. Res. 2, 189-194.

Robertson, A. (1962). Selection for heterozygotes in small populations. Genetics 47, 1291-1300.

Robertson, A. (1970). A theory of limits in artificial selection with many linked loci. In Biomathematics. Vol 1. Mathematical topics in Population Genetics. Ed. Kojima, K. Springer-Virlag, Berlin, pp. 246-288.

Robertson, F.W. (1954). Studies in quantitative inheritance V. Chromosome analysis of crosses between selected and unselected lines of different body size in Drosophila melanogaster. J. of Genet. 52, 494-520.

Robertson F.W. (1955). Selection response and the properties of genetic variations. Col Spring. Harb. Symp. Quant. Biol. 20, 166-177.

Robertson, F.W., (1957). Studies in quantitative inheritance XI. Genetic and environmental correlation between body size and egg production in Drosophila melanogaster. J. Genet. 50, 428-443.

Robertson, F.W. (1960a). The ecological genetics in Drosophila. 1. Body size and developmental time on different diets. Genet. Res. 1, 288-307.

Robertson, F.W. (1960b). 2. Selection for large body size on different diets. Genet. Res. 1, 305-318.

Robertson, F.W. (1960c). 3. Growth and competitive ability of strains selected on different diets. Genet. Res. 1, 303-350.

Robertson, F.W. (1963). 6. The genetic correlation between the duration of the larval period and body size in relation to larval diet. Genet. Res. 4, 74-92.

Robertson, F.W. and Sang, J.H. (1944). The ecological determinant of population growth in Drosophila culture. I. Fecundity of adult flies. Proc. Roy. Soc. B. 132, 258.

Robertson, F.W. and Reeve, E.C.R. (1952). Studies in quantitative inheritance. I The effects of selection on wing and thorax length in Drosophila melanogaster. J. Genet. 50, 414-448.

Robertson, F.W. and Reeve, E.C .R. (1955a). Studies in Quantitative inheritance III. Crosses between strains of different body size in Drosophila melanogaster. Zeitschriff für indukt. Abstammungs und Vererbungslehre, Bd. 86, 427-438.

Robertson, F.W. and Reeve, E.C.R. (1955b). VIII. Further analysis of heterosis in crosses between inbred lines of Drosophila melanogaster. Zeitschrift für indukt. Abstammungs und Vererbungslehre, Bd. 86, 439-458.

Roberts, R.C. (1966a). The limits to artificial selection for body weight in the mouse. I The limits attained in earlier experiments. Genet. Res. 8, 347-360.

Roberts, R.C. (1966b). II The Genetic nature of the limits. Genet. Res. 8, 361-375.

Sang, J.H. (1956). The quantitative nutritional requirements of Drosophila melanogaster. J.exp.Biol. 33, 45-72.

Sang, J.H. (1962). Selection for rate of larval development using Drosophila melanogaster cultured axenically on deficient diets. Genet. Res. 3, 90-109.

Sewell, D., Burnett, B. and Connolly, K. (1975). Genetic analysis of larval feeding behaviour in Drosophila melanogaster. Genet. Res. 24, 163-173.

Siegel, P.B. (1962). A double selection experiment for body weight and breast angle at eight weeks of age in chickens. Genetics 47, 1313-1319.

Spiers, J.G.C. (1974). The effect of larval competition on a quantitative character in Drosophila melanogaster. Ph.D. Thesis. University of Edinburgh.

Tantawy, A.O. (1956). Selection for long and short wing length in Drosophila melanogaster with different systems of mating. Genetics 28, 231-262.

Tantawy, A.O. (1957). Heterosis and genetic variance in hybrids between inbred lines of Drosophila melanogaster in relation to the level of homozygosity. Genetics 42, 535-543.

Tantawy, A.O. and Vetukhir, M.O. (1960). Effects of size on fecundity, longevity and viability in populations of Drosophila pseudoobscura. The American Nat. 94, 395-403.

Verghese, M.W. and Nordskog, A.W. (1968). Correlated responses in reproductive fitness to selection in chickens. Genet. Res. 11, 221-238.

Wallinga, J.H. and Bakker, H. (1978). Effect of long-term selection for litter size in mice on lifetime reproduction rate. J. Anim. Sci. 46, 1563-1571.

Wilson, S.P.H., Goodale, D., Kyle, W.H. and Godfrey, E.F. (1971). Long-term selection for body weight in mice. J. Heredity 62, 228-234.

Wright, S. (1931). Evolution in Mendelian populations. Genetics 16, 97-159.

Wright, S. (1938). Size of population and breeding structure in relation to evolution. Science 87, 430-431.

Wright, S. (1952). The genetics of quantitative variability Quantitative Inheritance, Ed. E.C.R. Reeve and C.H. Waddington. London: H.M.S.O. pp 5-41.

Wright, S. (1967). The foundations of population Genetics. In Heritage from Mendel. Ed. Brink, L.G. University of Wisconsin Press, 245-263.

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APPENDIX

TABLE 1 Selection differentials of BSM and BSH lines and their accumulative selection differentials at the limit. ($\Sigma\delta$)

Generations	BSM_1	BSM_2	BSM_3	BSM_4	$\overline{BSM}$	BSH_1	BSH_2	BSH_3	BSH_4	$\overline{BSH}$
1	2.91	3.32	2.75	1.89	2.71	5.54	5.18	5.14	5.60	5.36
2	2.26	2.58	1.96	1.86	2.16	4.73	3.90	4.14	3.39	4.04
3	1.70	2.53	1.92	2.33	2.12	3.87	3.47	3.64	3.63	3.65
4	2.04	2.22	1.68	2.11	2.01	4.33	4.60	3.59	3.29	3.75
5	2.29	2.00	2.55	2.32	2.24	3.73	4.60	2.94	3.43	3.67
6	2.16	1.90	2.18	1.00	1.96	3.50	3.30	3.31	3.72	3.44
7	1.96	2.37	2.89	2.14	2.32	2.36	2.75	4.95	4.22	3.57
8	1.88	1.69	1.30	3.60	2.12	1.67	3.39	4.15	3.07	3.07
9	1.71	2.35	2.13	1.87	2.01	2.54	3.37	2.10	3.39	2.85
10	1.46	1.64	1.18	1.95	1.55	2.02	2.36	2.43	2.34	2.28
11	.93	1.43	1.40	1.71	1.36	2.60	2.15	3.14	2.94	2.70
12	.62	1.47	1.71	1.47	1.37	3.17	1.90	2.64	1.98	2.42
13	1.39	1.07	1.43	1.63	1.38	2.42	2.46	2.49	2.07	2.34
14	1.25	.73	1.20	1.75	1.23	2.41	2.90	2.30	1.66	2.31
15	1.35	1.46	.85	1.60	1.31	2.25	2.26	2.15	2.16	2.20
16	1.43	1.40	1.09	1.54	1.36	3.19	1.95	2.53	2.32	2.49
17	1.57	1.43	1.10	.77	1.27	2.32	1.85	2.19	1.42	1.94
18	1.45	1.43	1.93	1.31	1.53	2.94	1.94	2.81	2.52	2.55
19	1.75	1.54	1.38	1.17	1.46	2.47	1.88	2.00	2.11	2.11
20	1.48	1.25	1.52	1.22	1.36	2.85	1.66	2.33	2.56	2.35
21	1.93	1.46	1.74	1.62	1.68	2.05	2.15	2.76	2.12	2.27
22	1.30	1.22	1.29	1.75	1.39	2.55	2.62	2.68	2.35	2.55
23	1.87	1.46	1.14	1.40	1.46	2.07	2.35	2.60	2.65	2.41
24	1.24	1.79	1.35	1.39	1.44	2.62	2.95	2.50	2.25	2.58
25	1.23	1.18	1.46	1.49	1.36	2.83	2.73	2.98	2.05	2.64
26	1.53	1.63	1.32	1.54	1.50	3.75	3,13	2.58	2.29	2.93
27	1.14	1.98	1.14	1.25	1.37	2.61	2.63	2.66	2.33	2.55
28	1.01	1.55	1.39	1.11	1.26	2.88	2.68	2.83	2.09	2.62
29	1.17	1.28	1.04	1.25	1.18	2.83	2.27	2.32	2.92	2.58
30	1.21	1.33	0.94	1.37	1.21	3.03	3.07	2.66	2.63	2.84
31	1.00	1.21	1.30	1.30	1.22	3.35	3.22	2.10	3.03	2.92
32						2.97	2.69	2.44	3.71	2.95
33						3.14	2.79	2.27	3.19	2.84
34						3.21	3.00	2.30	2.26	2.94
35						2.49	2.42	2.24	2.65	2.45
36						3.02	2.39	2.38	2.76	2.63
$\Sigma\delta$	47.21	50.90	48.35	51.32	50.78	106.3	100.9	101.1	100.1	101.9
$\Sigma\delta/\sigma$	14.84	16.00	15.20	16.13	15.96	33.42	31.7	31.7	31.4	32.0

TABLE 2 Selection differential of BLM and BLH lines and their accumulative selection differential at the limit.

Generations	BLM_1	BLM_2	$\overline{BLM}$	BLH_1	BLH_2	$\overline{BLH}$
1	3.27	2.74	3.00	6.02	6.05	6.03
2	2.19	2.48	2.33	4.20	3.67	3.93
3	2.04	2.28	2.11	3.95	3.85	3.90
4	2.01	2.21	2.11	4.11	3.91	4.01
5	2.17	2.10	2.13	4.14	4.09	4.11
6	2.73	2.30	2.51	6.23	3.80	5.01
7	1.45	2.09	1.77	5.43	3.26	4.34
8	1.69	1.71	1.70	3.28	3.28	3.28
9	1.77	1.60	1.68	3.64	2.14	2.89
10	1.54	1.55	1.54	2.02	2.51	2.26
11	1.48	1.89	1.68	2.93	2.96	2.94
12	1.91	1.27	1.59	2.52	2.41	2.46
13	1.35	1.43	1.39	2.25	2.84	2.54
14	1.35	1.15	1.25	2.44	2.53	2.40
15	1.47	1.68	1.57	3.02	3.85	3.43
16	1.65	1.95	1.80	2.54	2.51	2.52
17	1.58	1.63	1.60	1.80	3.63	2.71
18	1.03	1.47	1.26	2.18	2.58	2.38
19	1.28	1.86	1.57	1.54	2.65	2.09
20	1.44	1.36	1.40	2.22	2.73	2.47
21	1.58	1.38	1.48	3.33	2.86	3.09
22	1.26	1.27	1.26	2.90	2.85	2.87
23	1.49	1.49	1.49	2.77	3.21	2.99
24	1.95	1.35	1.65	2.30	3.84	3.07
25	1.66	1.37	1.51	2.80	3.02	2.91
26	1.72	2.19	1.91	2.70	3.07	2.88
27	1.71	1.42	1.56	3.21	3.44	3.32
28	1.51	1.97	1.78	2.44	2.99	2.71
29	1.60	1.64	1.69	2.62	3.08	2.85
ΣS	48.36	50.73	49.54	91.53	93.58	92.55
$\Sigma S/\sigma$	15.20	15.95	15.57	28.78	29.42	29.10

TABLE 3 Selection differentials of pupae lines and their accumulative selection differentials at generation 20.

	PSM_1	PSM_2	PSM_3	PSM_4	$\overline{PSM}$	PSH_1	PSH_2	PSH_3	PSH_4
1	15.16	12.25	20.76	14.95	15.78	22.5	17.89	21.46	16.84
2	23.15	13.20	18.00	9.75	16.02	32.02	23.42	28.61	22.98
3	14.75	9.50	13.00	11.80	12.06	25.19	23.37	21.02	24.81
4	13.36	13.20	12.69	5.00	11.05	31.96	20.93	27.37	19.70
5	16.05	13.10	9.45	8.05	11.66	19.05	23.50	31.14	19.71
6	12.49	4.50	5.49	5.00	12.49	21.58	18.80	9.99	9.70
7	18.20	10.25	11.50	23.35	15.82	15.42	23.74	16.67	19.50
8	8.45	11.25	11.80	7.95	9.86	14.87	11.55	18.77	21.65
9	16.06	8.85	9.85	6.87	10.40	20.13	13.41	20.82	16.30
10	8.40	9.50	15.05	12.15	11.27	20.72	17.10	17.09	19.14
11	10.80	9.35	6.80	10.51	9.26	16.10	18.43	15.54	15.36
12	14.30	8.90	12.45	8.50	11.01	18.66	16.09	23.05	26.06
13	13.85	10.00	10.63	10.95	11.35	18.98	26.40	15.98	18.12
14	17.20	9.60	11.10	10.56	12.11	20.74	15.70	18.44	14.25
15	12.35	17.30	9.27	3.35	10.55	20.18	18.76	15.78	15.06
16	11.05	9.10	6.65	11.18	9.54	19.56	15.04	17.70	17.77
17	9.65	11.50	6.90	7.45	8.87	19.29	20.58	19.98	19.09
18	12.40	9.65	12.10	14.89	12.26	16.46	15.96	13.54	19.09
19	15.03	11.70	8.45	8.00	10.80	14.18	18.58	20.66	17.09
20	7.05	9.55	8.90	8.40	8.47	14.51	17.38	16.96	17.44
$\Sigma\delta$	270.02	212.25	220.84	198.66	230.63	402.10	376.58	390.67	369.65
$\Sigma\delta/8$	13.23	10.38	10.80	9.71	11.28	19.67	18.42	19.11	18.08

(TABLE 3 Continue ...)

	$\overline{PSH}$	PLM_1	PLM_2	$\overline{PLM}$	PLH_1	PLH_2	$\overline{PLH}$
1	19.67	11.21	10.58	10.89	23.71	34.57	29.14
2	26.75	15.99	12.98	14.48	26.95	24.07	25.51
3	23.59	12.36	17.64	15.00	27.75	27.84	27.79
4	24.99	13.81	12.25	13.03	32.89	18.26	25.57
5	23.35	18.21	11.99	15.10	21.81	20.12	20.96
6	20.19	9.94	4.32	7.13	27.34	32.33	29.83
7	18.83	9.19	10.31	9.75	25.89	26.45	26.17
8	16.71	7.93	8.71	8.32	16.08	13.12	14.60
9	17.66	9.72	9.06	9.39	10.05	18.12	14.08
10	18.51	8.29	8.30	8.29	14.73	25.01	19.91
11	16.35	9.85	13.07	11.77	20.74	19.14	19.94
12	20.96	9.74	11.76	10.75	15.93	23.79	19.86
13	19.87	12.14	8.85	10.49	18.25	18.07	18.16
14	17.28	9.62	8.95	9.28	15.05	19.34	17.19
15	17.44	8.57	11.37	9.97	16.35	18.84	16.09
16	17.38	11.77	11.04	11.40	14.84	18.73	16.78
17	19.73	9.78	8.65	9.21	14,11	19.37	16.74
18	16.26	10.80	11.79	11.29	15.10	19.69	17.39
19	17.61	8.90	10.27	9.58	15.37	16.79	16.08
20	16.56	7.06	8.69	7.87	13.59	14.49	14.04
$\Sigma\delta$	384.75	214.87	210.58	212.97	386.53	428.14	405.83
$\Sigma\delta/\sigma$	18.82	10.51	10.30	10.41	18.90	20.94	19.85

TABLE 4 Genetic correlation estimates of body length and pupae number in the 5th, 10th and 20th generations of pupae lines.

	GENERATIONS		
Lines	5th	10th	20th
PSC_1	-4.08 ± 36.8[+]	.375 ± 1.12	-20.34 ± 26.7
PSC_2	-2.65 ± 4.52	-3.08 ± 8.9	-.158 ± .33
PSC_3	-.447 ± .297	-.318 ± .33	3.19 ± 24.7
PSC_4	-.161 ± .380	.112 ± .42	
$\overline{PSC}$	-2.25 ± 3.62	-.734 ± .20	-.499 ± .327
PSM_1	0.163 ± .479	-.163 ± .44	-.829 ± .108
PSM_2	-2.97 ± 37.2	.082 ± .75	
PSM_3	-.727 ± .132	.830 ± .18	.327 ± 4.12
PSM_4	-.916 ± .062	.041 ± .36	
$\overline{PSM}$	-.374 ± .264	.200 ± .673	-.155 ± .32
PSH_1	-.241 ± .788	-.086 ± .49	-1.33 ± .416
PSH_2	.190 ± .468	-.784 ± .61	3.39 ± 5.24
PSH_3	.161 ± .599	-8.69 ± 47.6	
PSH_4	-1.480 ± .848	-1.28 ± .45	
$\overline{PSH}$	-.094 ± .535	-.664 ± .174	
PLC_1	-.234 ± .603	-.153 ± 1.37	1.62 ± 1.2
PLC_2	-.975 ± .049		-.003 ± .08
$\overline{PLC}$	-.490 ± .128		1.18 ± 1.01
PLM_1	.073 ± .712	-2.14 ± 4.00	
PLM_2	-5.14 ± 5.4	.183 ± 1.4	
$\overline{PLM}$	-4.45 ± 4.2	-.855 ± .161	
PLH_1		.172 ± .503	-.82 ± .16
PLH_2	-.059 ± .795	-.445 ± .296	3.81 ± 1.1
$\overline{PLH}$		-.098 ± .110	-1.688 ± .92

[+] Standard errors were calculated following Hill (1971).

TABLE 5 Genetic correlation estimates of body length and pupae number in generation 5, 10 and at the limit of body lines.

	GENERATIONS		
Lines	5	10	Limit
BSC_1	-.098 ± .231[+]		
BSC_2	.175 ± .15	-1.230 ± .87	.280 ± .21
BSC_3	.359 ± .35	.595 ± .35	-.140 ± .43
BSC_4	-.265 ± .54		-.180 ± .39
$\overline{BSC}$	.136 ± .08	-.301 ± .18	.026 ± .75
BSM_1	-2.290 ± .29	1.130 ± .15	-.679 ± .04
BSM_2	-.896 ± .42	.600 ± .12	
BSM_3	-1.680 ± .56	.740 ± .10	.847 ± .12
BSM_4	.440 ± .49	.650 ± .06	
$\overline{BSM}$	-1.230 ± 1.00	1.650 ± 1.92	.380 ± .138
BSH_1	-.151 ± .62	-4.20 ± .53	-.740 ± .08
BSH_2	-.081 ± .29	-.320 ± .18	
BSH_3	-.392 ± .163	-.570 ± .27	
BSH_4	-.420 ± .48	.161 ± .42	
$\overline{BSH}$	-1.980 ± .12	-.952 ± .25	
BLC_1	-.966 ± .63	-1.090 ± 1.67	
BLC_2	.156 ± .29	-.587 ± .13	-.736 ± .07
$\overline{BLC}$	-.090 ± .15	-.770 ± .07	
BLM_1	-1.940 ± .34	-.290 ± .73	
BLM_2	-3.600 ± .45	-1.190 ± .39	.860 ± .32
$\overline{BLM}$	-2.600 ± 1.70	-1.080 ± .48	
BLH_1	-1.340 ± .28	3.500 ± .44	
BLH_2	-.161 ± .20	-2.030 ± .27	.340 ± .35
$\overline{BLH}$	-.692 ± .05	1.950 ± .18	

[+] Standard errors were calculated following Hill (1971).

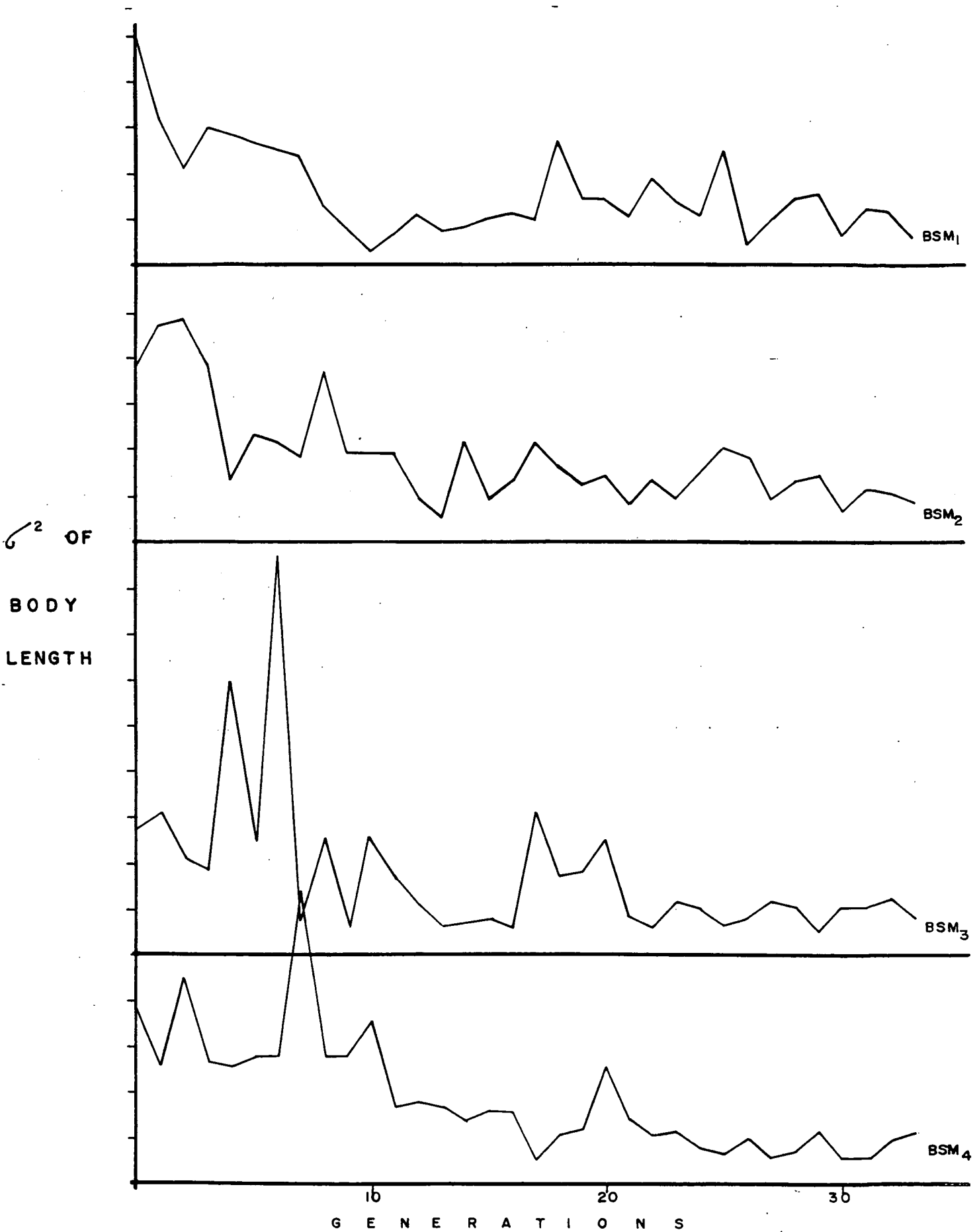

FIG. 1 CHANGE OF PHENOTYPIC VARIANCE OF BSM LINES

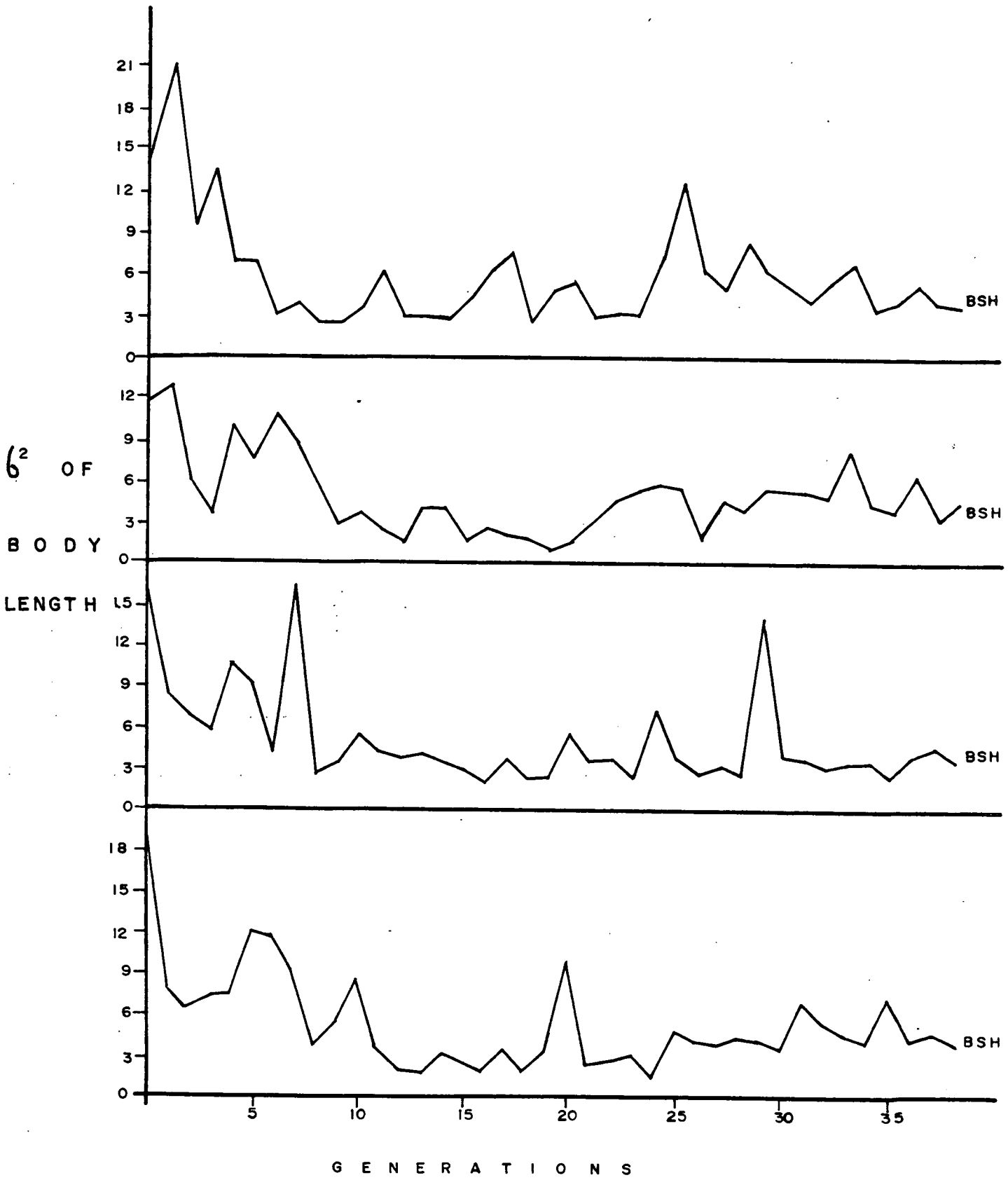

FIG.2 CHANGE OF PHENOTYPIC VARIANCE OF BSH LINES

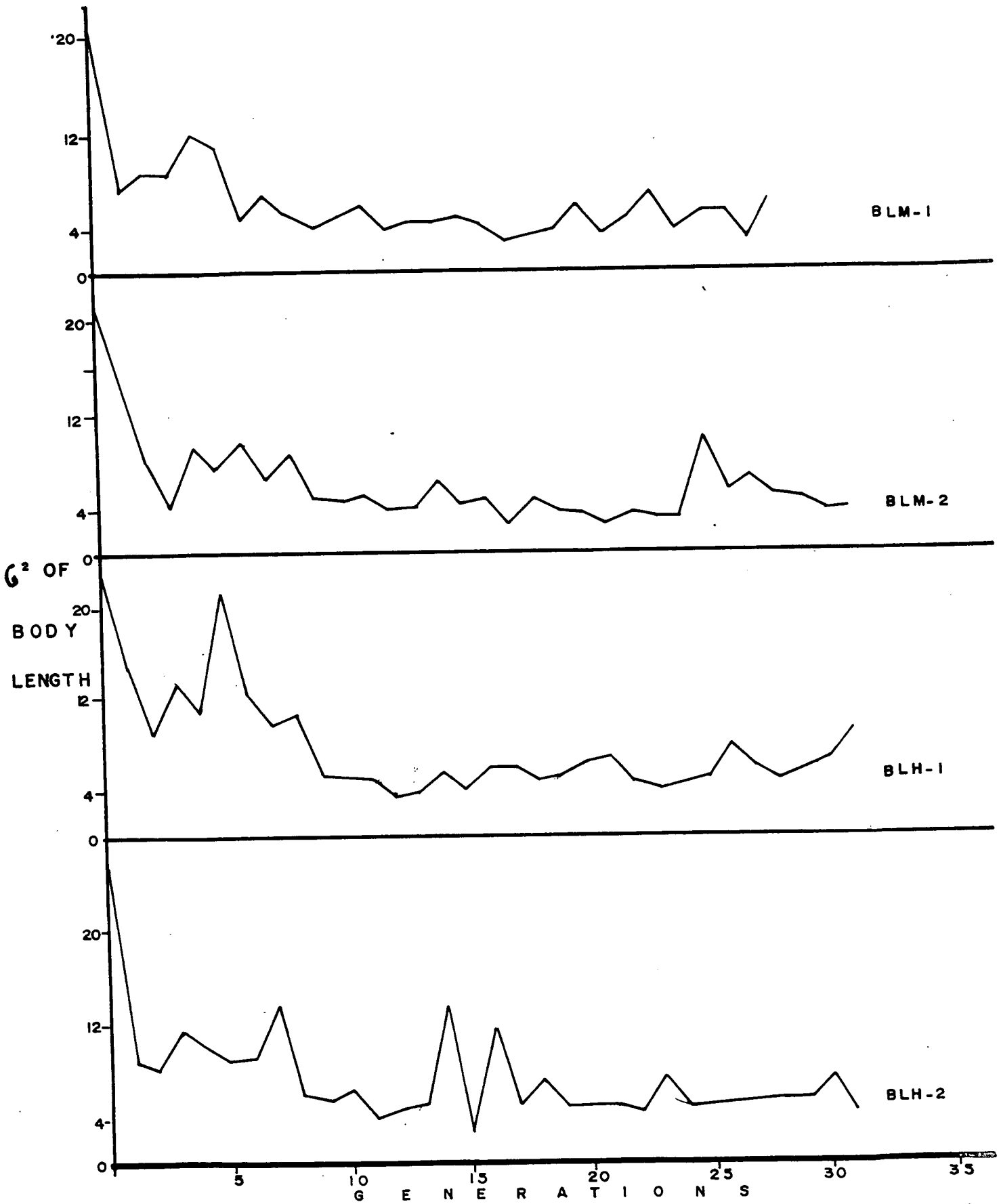

FIG.3 CHANGE OF PHENTYPIC VARIANCE OF BLH AND BLM LINES

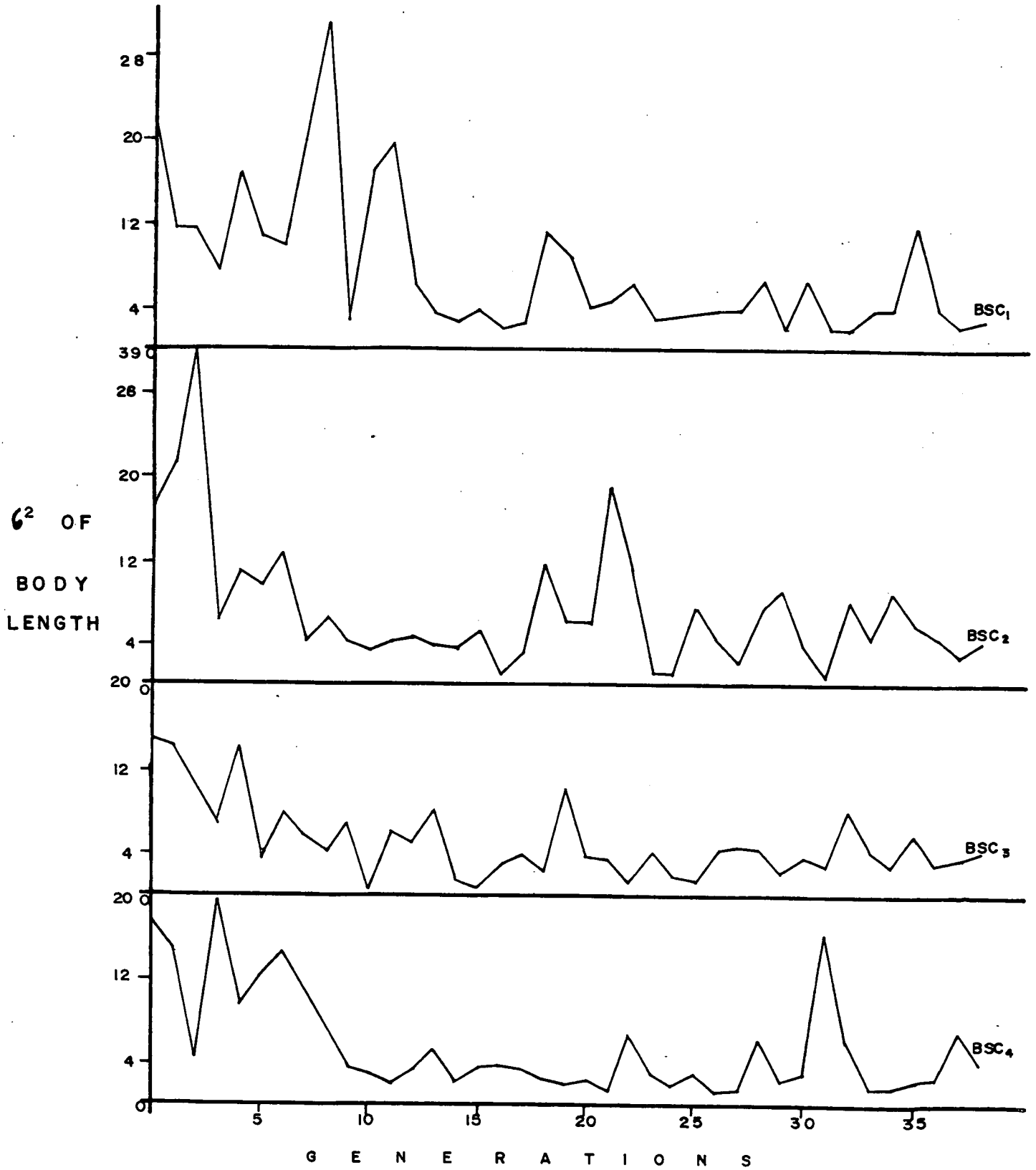

FIG. 4 CHANGE OF PHENOTYPIC VARIANCE OF BSC-LINES

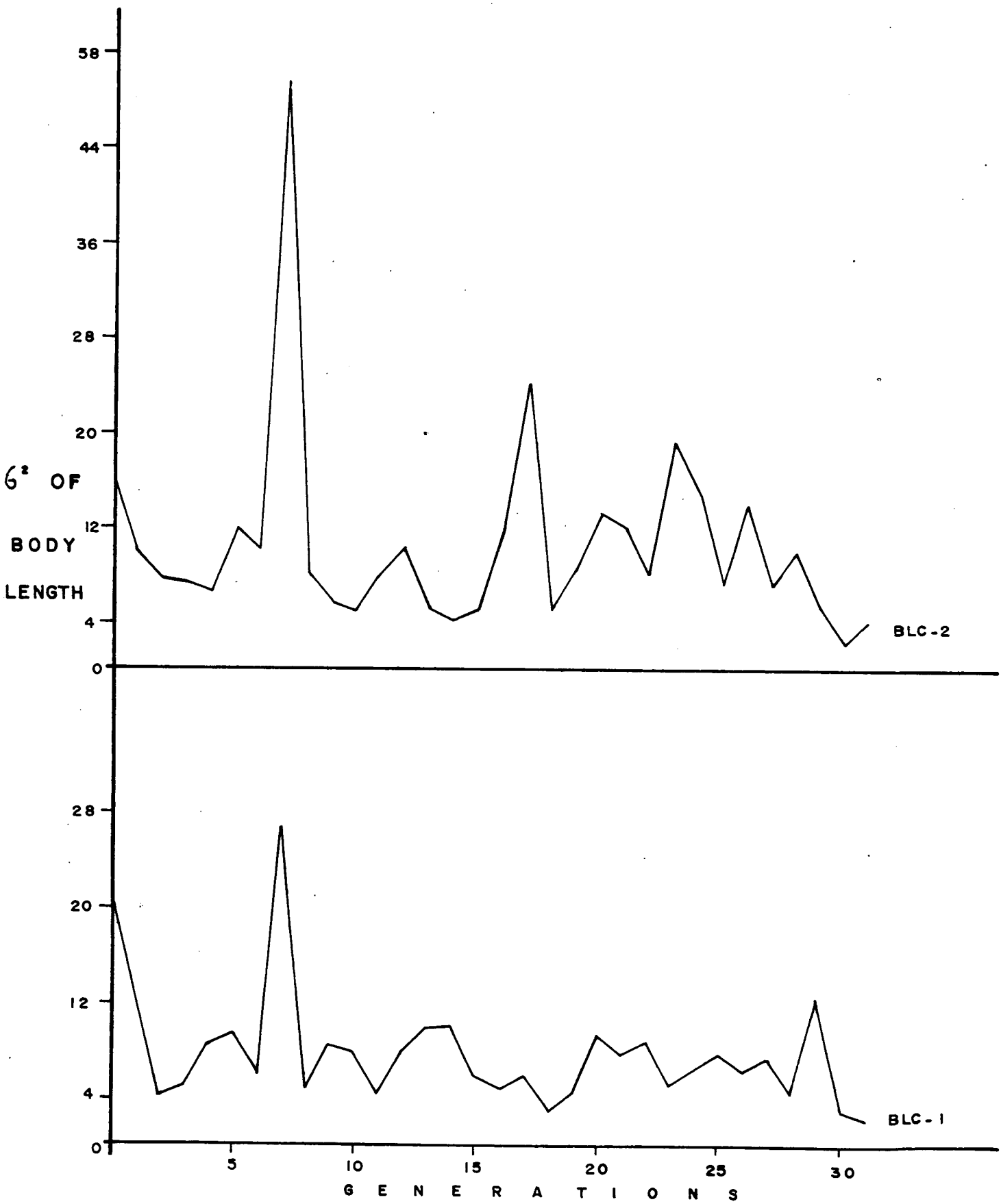

FIG.5 CHANGE OF PHENOTYPIC VARIANCE OF BLC LINES

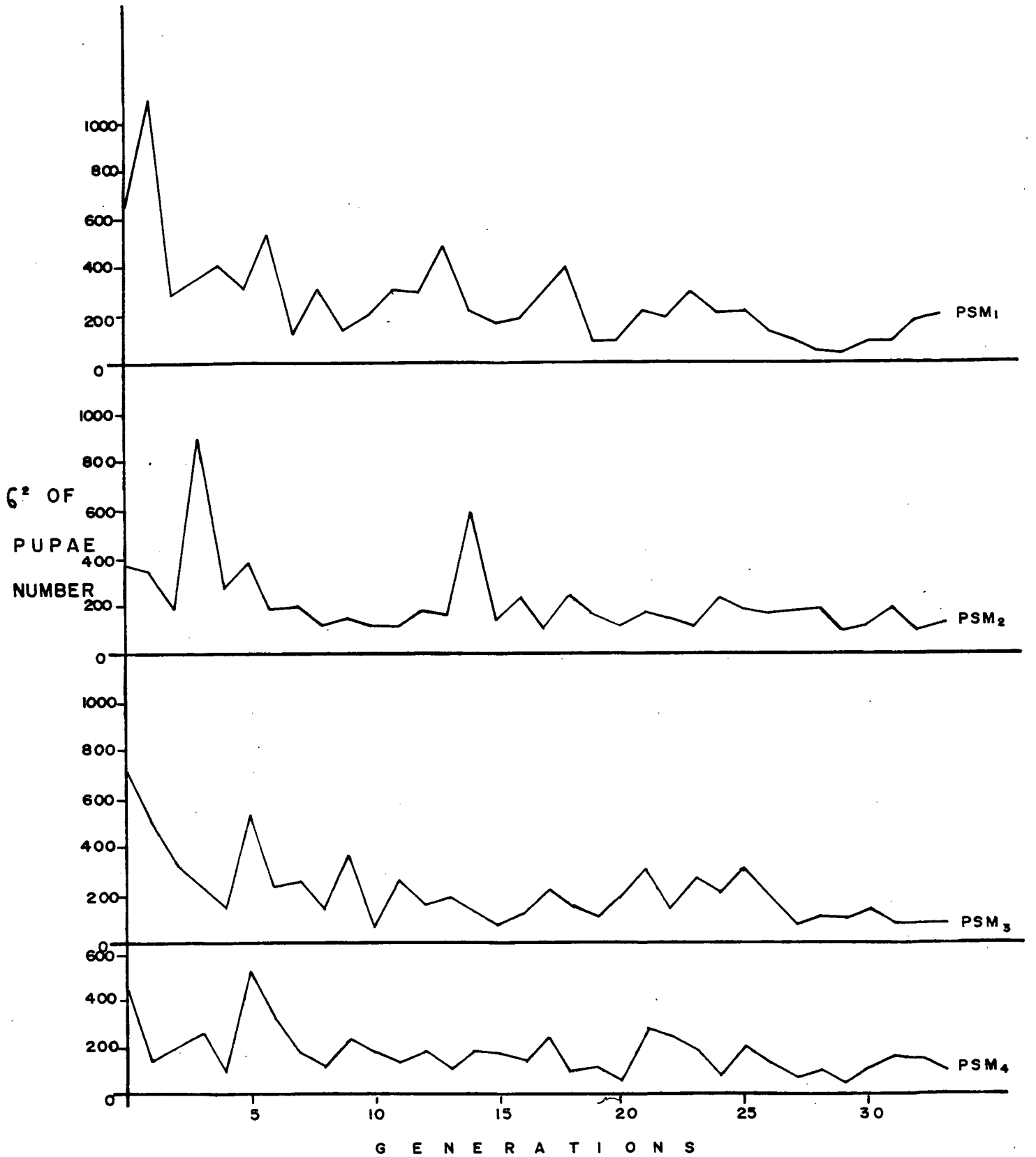

FIG. 6 CHANGE OF PHENOTYPIC VARIANCE OF PSM LINES

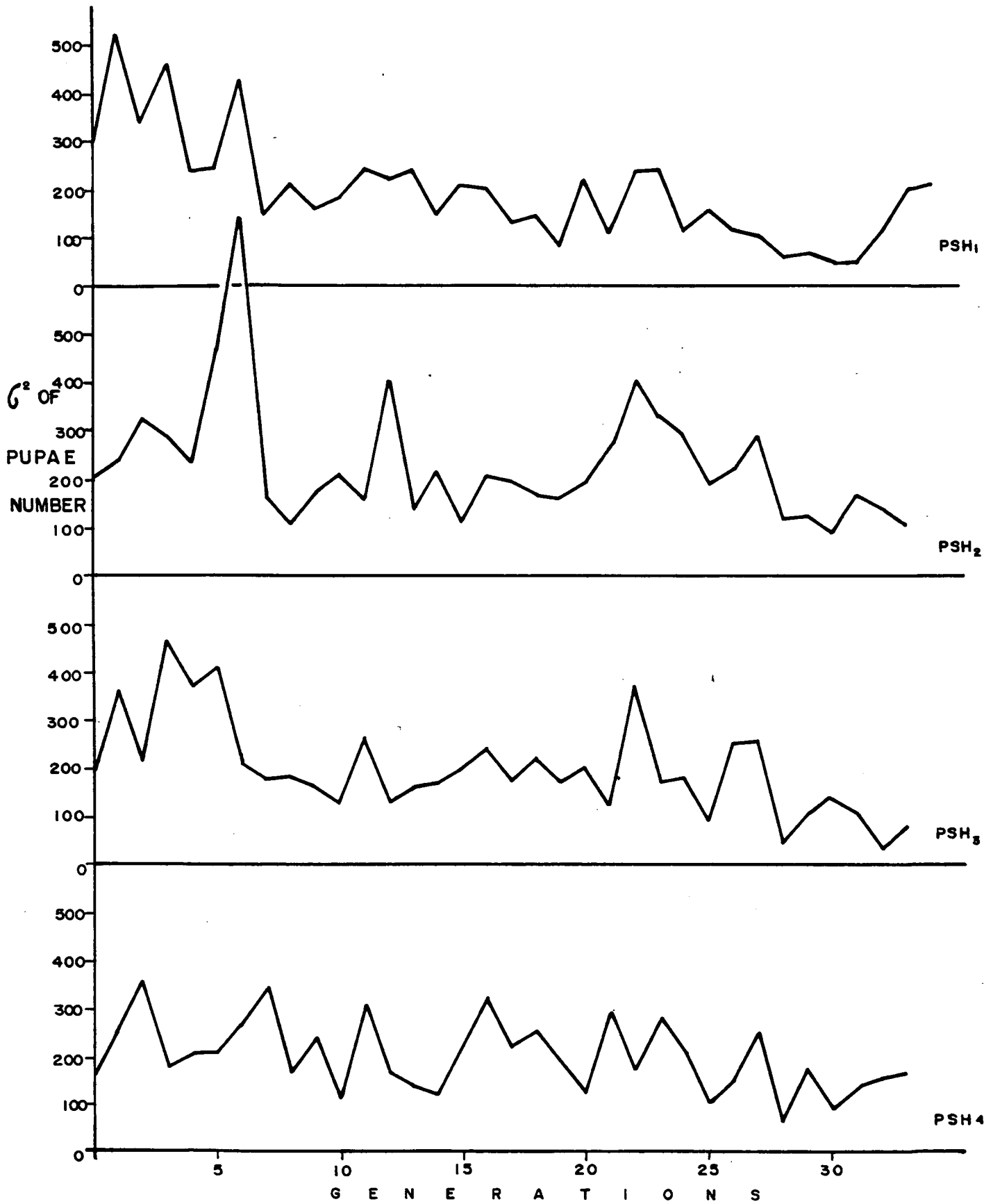

FIG. 7 CHANGE OF PHENOTYPIC VARIANCE OF PSH LINES

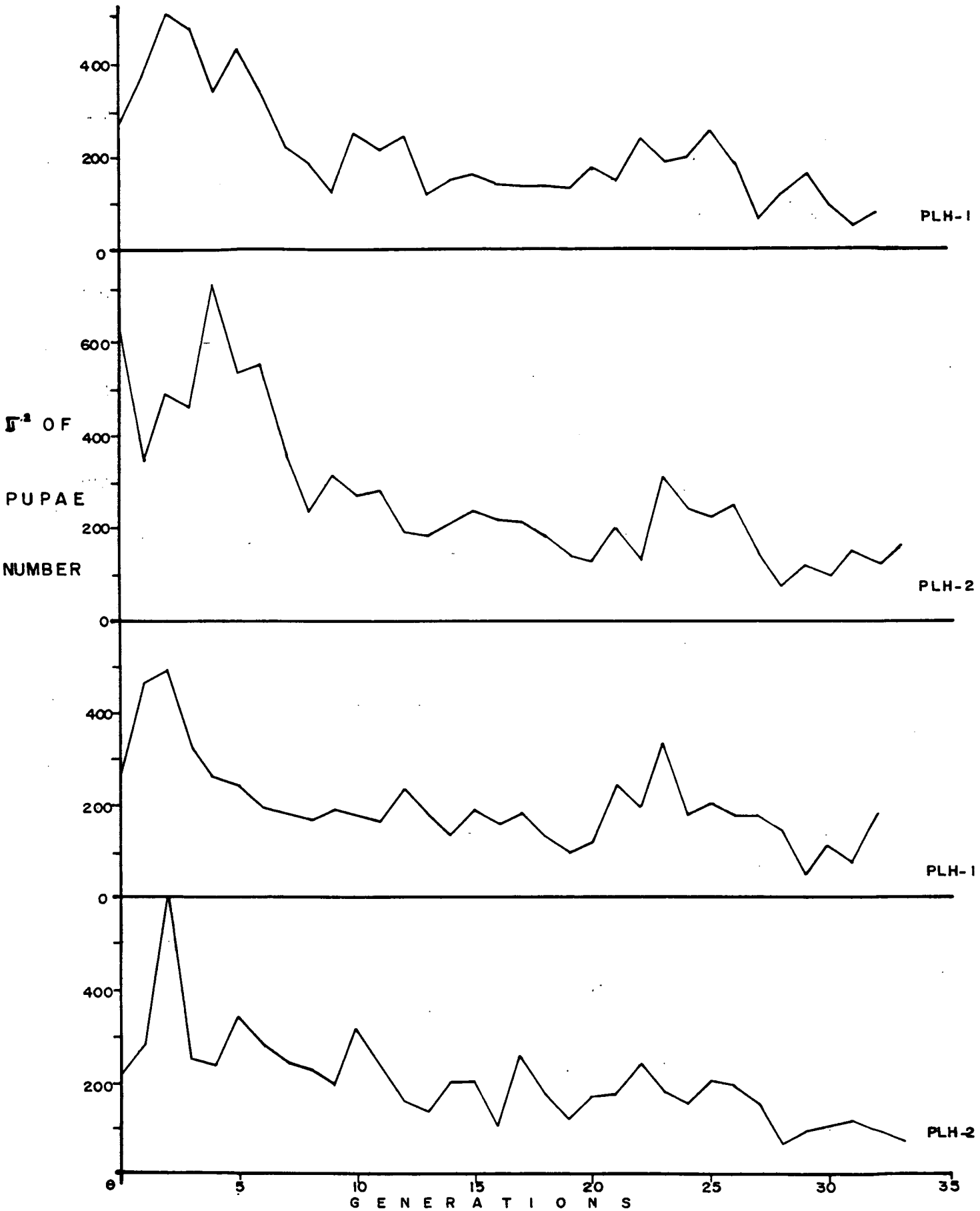

FIG 8 CHANGE OF PHENOTYPIC VARIANCE OF PLM AND PLH LINES

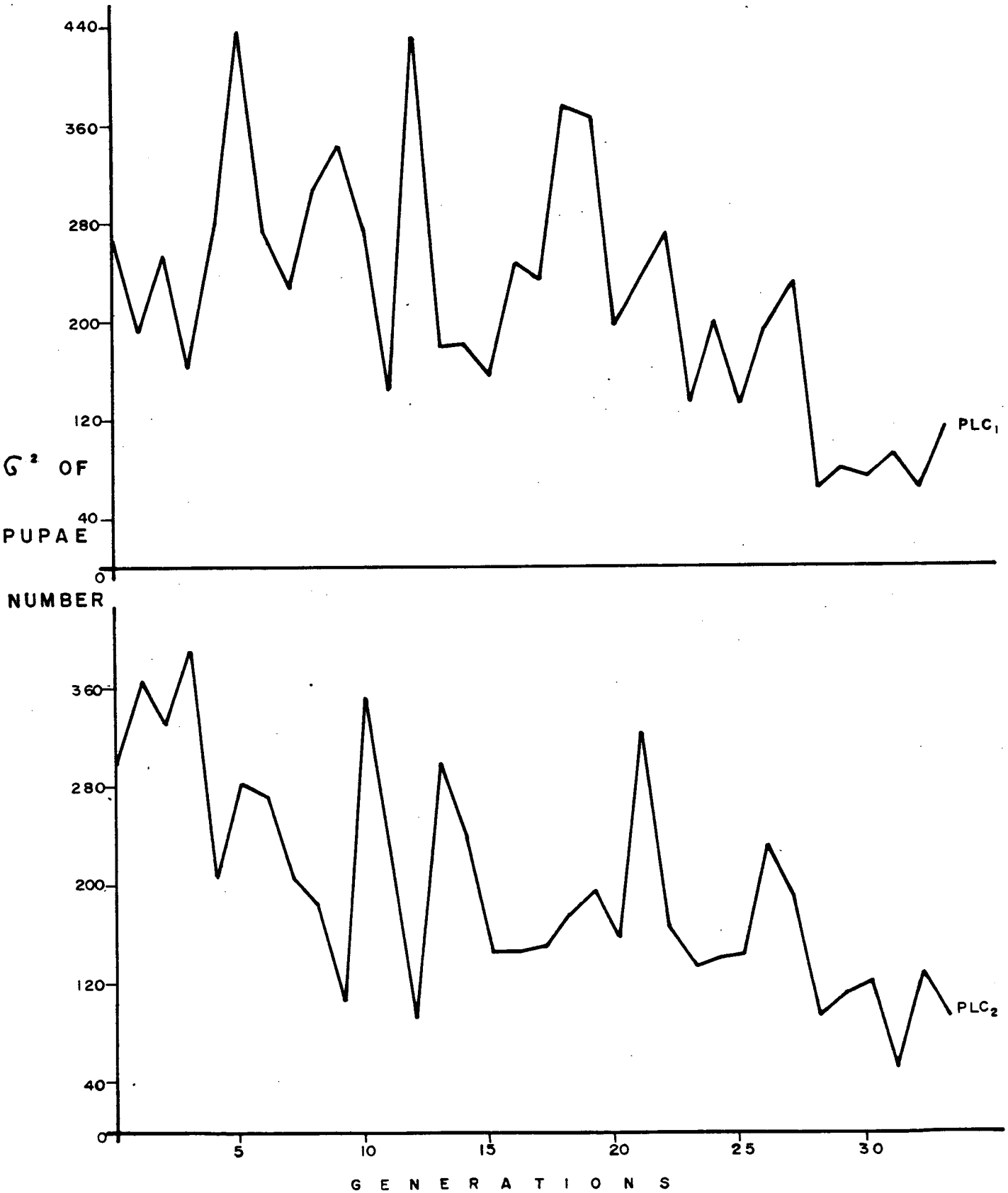

FIG. 9 CHANGE OF PHENOTYPIC VARIANCE OF PLC LINES

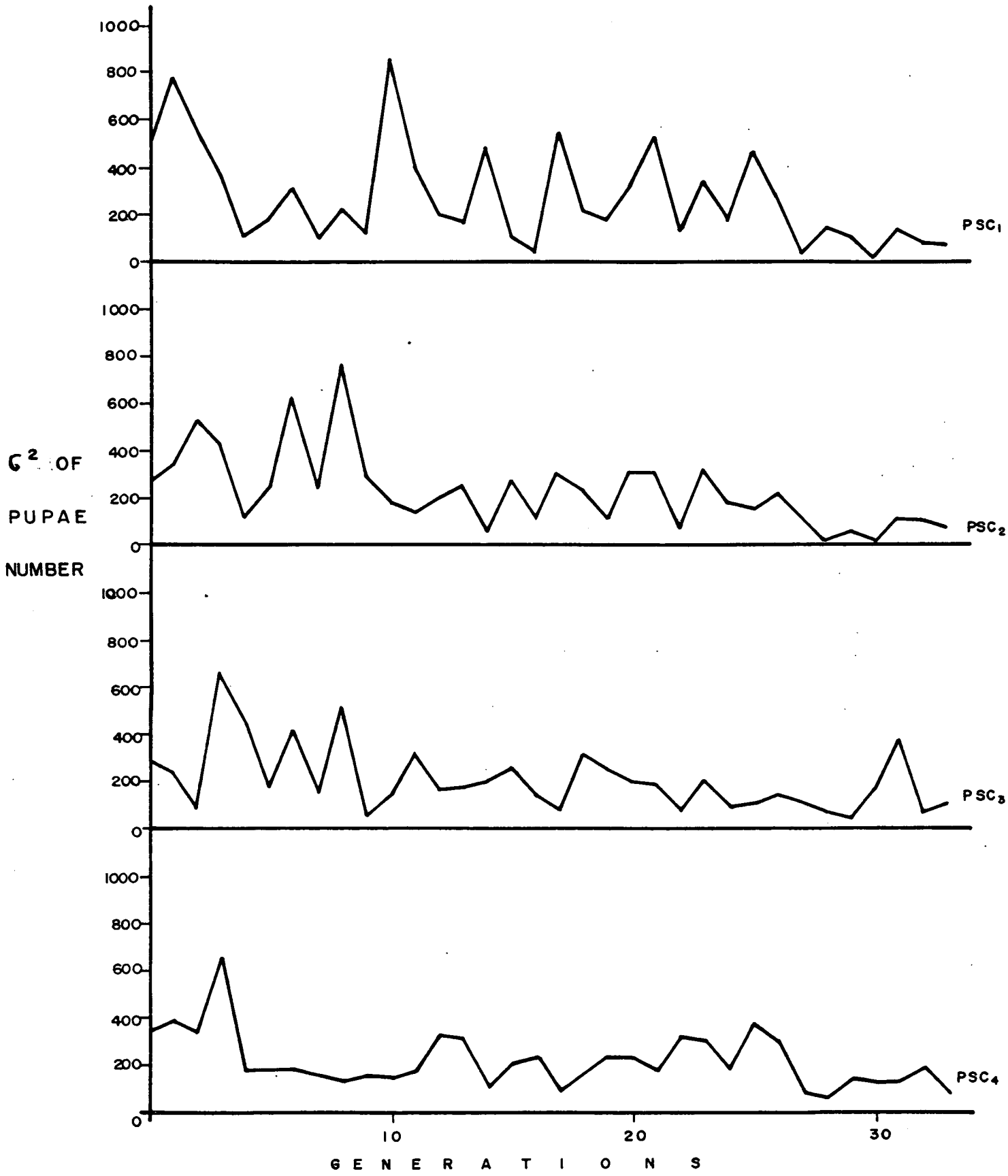

FIG.10 CHANGE OF PHENOTYPIC VARIANCE OF PSC LINES